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AN

# ANATOMICAL DESCRIPTION

OF

# THE DISEASES

OF THE ORGANS OF

# CIRCULATION AND RESPIRATION.

BY

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# EDITOR'S PREFACE.

The following pages will not be found to contain a mere descriptive catalogue of curiosities in morbid anatomy, nor records of extreme or severe cases only, but a thorough anatomical and physiological account of the origin of disease, of its progress through its several phases, and of its ultimate issue in death, in abiding organic mischief, or in recovery. The practical utility of this plan, apart from the truthfulness and ability with which it is carried out, few will be disposed to contest; for, to use the words of a highly distinguished physician of the present day,[1]—" so far as morbid anatomy contemplates the last or latest results of disease that are fixed and irremediable, and unalterable, its value is very small. But so far as morbid anatomy contemplates *disease in progress,* and scrutinizes and explains its organic processes, its value is very great."

My former fellow-student and, I have both pride and pleasure in adding, my intimate friend, Professor E. Hasse, conceived a very early predilection for pathological anatomy, and this bias was fostered and matured by a lengthened sojourn at the schools and hospitals of Paris and of Vienna. On his return to his own university, Leipsic, he was appointed by

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[1] Dr. P. M. Latham.

[illegible]4801[illegible]

my revered clinical instructor, Professor Clarus, to be assistant clinical teacher, and also pathological prosector at the principal hospital. With such means at his disposal, my friend forthwith commenced forming a pathological collection, which, under his auspices, has grown into a most interesting and valuable museum; and the present work is but a collateral result of the unwearied *practical industry* which he displayed in that undertaking, combined with a thorough knowledge of all that other observers had before achieved in the same field of science.

Hence it will at once be seen that this treatise differs essentially from what is commonly called a compilation. The high estimation in which it is held in Germany, is clearly shown by the fact that, since its publication, Professor Hasse has had the offer of the chair of clinical medicine from no fewer than five universities. He has accepted that vacated by Professor Schönlein, at Zurich, and at present holds the additional rank of Rector of that Germano-Swiss University.

When the Council of the Sydenham Society did me the honour to authorize me to translate Professor Hasse's work, they, at the same time, suggested that I should first request my friend to revise the original, published several years previously. This duty he willingly undertook, and has faithfully and carefully fulfilled, the result being that, whilst some chapters have been but little altered in this revision, others,—for example, that on the Diseases of the Heart,—may be said to have been entirely rewritten. Much additional information has also been thus furnished concerning the microscopic characters of a variety of diseases. In short, the translation is to be looked upon as a completely new edition of the book.

The original is intended by the author as the first of a series of tomes, comprising the diseases of every system and organ of the body. But the uncertainty that necessarily attaches to the appearance of comprehensive works in distinct parts, has induced the Council to prefer publishing the present volume, which constitutes singly a complete and valuable treatise, as a separate and independent work. This has, however, rendered it necessary to omit a portion of the general preface, not altogether applicable to the following pages. In the preparation of the translation I have been favoured with the assistance of Mr. A. Ure, who, at the request of the Council, has revised the MS.; and I have, moreover, to offer my grateful acknowledgments to the Secretary of the Society, Dr. J. Risdon Bennett, for his obliging courtesy to me on all occasions, as well as for the able and judicious aid and advice which he has afforded me in my capacity as editor.

*Foley Place, Feb.* 1846.

# AUTHOR'S PREFACE.

In composing the following work, the author's aim has been to make the actual knowledge of Pathological Anatomy subservient to an *Anatomical History of Disease;* and to attain this object the more fully, he has not relied solely upon his own investigations, but also largely availed himself of facts recorded by others. In making use of other men's experience, he has, however, found it necessary to be extremely circumspect, and, in order to avoid falling into serious error, has been obliged to disregard many observations as being imperfectly reported. Carelessness in describing the results of an examination of the dead body is frequent, and, in such cases, it is unfortunately too common to substitute opinions for facts. Thus an organ is simply stated to be "inflamed," without any attempt being made to describe it by the characters that distinguish inflammation from other preternatural conditions,—although to discriminate between them is not always an easy matter. The results of pathological dissections would doubtless prove infinitely more serviceable to science, if these were generally consigned to skilful hands. A mere student in medicine is frequently employed in an office for which even a perfect physiological anatomist might scarcely be deemed qualified; and how often is the hasty examination of less than a single hour thought sufficient to solve the mysteries attendant upon a disease,—the growth of years. Nevertheless, the author has not failed to avail himself of the rich materials afforded by literature, so far as they were accessible to him; nor duly to acknowledge the sources from whence he has thus drawn.

Still all that has been hitherto achieved is too imperfect, and the means at the author's disposal have been too limited to allow of uniformity in the treatment of the various chapters of the present work. It is, therefore, to be looked upon for the most part, as a collection of monograph sketches, the working up of which must be left to future years and more able hands. Meanwhile the author's endeavour has been at least to adhere rigidly to simple observation, and to award to facts their full weight, rejecting all theory and hypothesis based upon a less solid foundation.

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# PART FIRST.

# DISEASES
OF THE
# ORGANS OF CIRCULATION.

# PATHOLOGICAL ANATOMY.

## CHAPTER I.

## DISEASES OF THE LYMPHATIC VESSELS AND GLANDS.

### SECT. I.—ANGIOLEUCITIS, LYMPHANGIOITIS, LYMPHADENITIS. INFLAMMATION OF LYMPHATIC VESSELS.

The lymphatic vessels, owing to their general and dense distribution within as well as between all the organs of the body, necessarily take part in every inflammation. From their simple structure and their uniform reticular ramifications, they can rarely attain a distinct individuality, or be so substantially isolated from the parenchyma of organs, or even from cellular tissue, as to escape, with the same frequency as arteries and veins, the influence of organic changes in the parts to which they belong. It is, therefore, only in unison with general experience, that Sömmering insists upon this participation of the absorbents in the inflammation of every part of the frame.[1] It would seem more important to demonstrate anatomically that the lymphatic vessels may become the primary seat of an inflammatory process.[2]

In the blood-vessels inflammation manifests its principal phenomena within the canals, whilst its consequences extend, especially in the instance of veins, to the remotest ramifications of the circulating system. In the lymphatic vessels the inflammatory process is originally developed in the contiguous cellular tissue, and from thence communicated, in a reflex manner, to the parietes of the canals. Again, the vitiated contents of individual branches of the lymphatic vessels do not at once go into the larger trunks, and from thence into the general circulation, but have previously to pass through a series of glands, following each other at various intervals, and seeming to operate, to a certain extent, like purifying instruments. Hence the consequences of a general diffusion of the morbid product, so serious in phlebitis, are here rarely to be apprehended.

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[1] De morbis vasorum absorbentium corp. hum. Traj. ad M. 1795.

[2] See Assalini, Cruikshank, and Gendrin (anatomical description of inflammation.) See also Tonnelé (Archives générales de Méd. 1830, vol. xxii), Duplay (Arch. gén. 1835, nouv. sér. vol. vii), Velpeau (Arch. gén. 1835, nouv. sér. vol. viii), Weitenweber (Beyträge, vol. iv, fasc. ii, 1839), Breschet (le Système lymphatique, &c., Paris, 1836, p. 268.)

It is likewise well deserving of consideration, that the lymphatic vessels being exclusively devoted to the function of absorption, can contain fluids of very various, even of morbid admixture, without detriment to their simple internal membrane; and that it is not until the fluids in question have reached the lymphatic glands, and have actually undergone a process of organic assimilation, that inflammatory reaction becomes established. This is particularly evident, when miasmatic or contagious matter is conveyed through the lymphatic system, as for instance, in typhus, in the plague, &c., where the lymphatic vessels never exhibit any morbid alteration, although the glands are found swollen, softened, and disorganized, if not wholly destroyed. In jaundice, from closure of the biliary ducts, Sömmering detected fluid of the colour of bile in the lymphatic vessels, without anything anormal in their structure. This applies of course only to the smaller branches, for owing to its larger caliber, the fuller development of its coats (some anatomists assign to it three), the more uniform nature of its contents, and its immediate junction with the left subclavian vein, the thoracic duct manifests, when diseased, tendencies similar to those of veins.[1]

From the foregoing introductory remarks, it may be inferred that the membranous canals of the lymphatic vessels never become the seat of idiopathic inflammation, but are either involved in the inflammatory affection of the surrounding textures, or else influenced by the vitiated condition of the contained fluid. The latter point chiefly concerns us here.

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[1] Vide Andral (Arch. gén. vol. vii, p. 502), and Sir Astley Cooper (Med. Records and Researches, vol. i, p. 87).

Having already shown the slight susceptibility of the lymphatic vessels to irritation, and, as resulting therefrom, the comparatively rare occurrence of genuine inflammation of their tunics, it remains for us to inquire under what circumstances a septic inflammation is brought about. Velpeau assumes a peculiar disposition. A first condition, however, is that of a liability to transudation of the morbid contents through the parietes of the lymphatic vessels. An exosmosis of this kind is induced, partly by the immediate action of the irritative matter upon the membranes of the vessels, partly by the clogging and distension of the latter, in proportion as the lymphatic glands become less permeable to the affluent lymph. This is abundantly confirmed by facts: for, during life, we observe that the lymphatic glands become turgid, whilst the course of the lymphatic vessels is indicated by a network of flat red stripes. After death, the surrounding cellular texture is found to be the principal seat of the inflammatory changes. This texture is saturated with the transuding fluid, which, at an early period, is limpid, and of a pale red colour, but afterwards becomes turbid,—either coagulating or being converted into pus. In the midst of this, the lymphatic vessels may be recognized as thin knotty cords; their coats being thickened, especially in the vicinity of the valves, and about the point of decussation of the individual branches. Externally they are milk white; their internal membrane, however, is of a pale red, and no longer smooth, but of a fine velvety aspect.

When suppuration begins to spread through the cellular tissue, the lymphatic vessels implicated become destroyed, and the whole is resolved into abscesses. These abscesses do not extend lengthwise, as in phlebitis, but form small, circumscribed and flattish accumulations, especially where the inflammation affects the superficial lymphatic vessels ramifying beneath the skin. When the deep-seated layer is attacked, the plexuses running in the course of the larger blood-vessels and nerves, are changed as above described. The cellular sheath of such vessels is then found thickened, infiltrated with lardaceous serum, and here and there destroyed by suppuration.

These inflammatory phenomena commonly extend to the next group of glands, which either sets a limit to the progress of the disease, or else becomes engaged in the inflammatory and suppurative processes, and offers a fresh starting-point for the disease in the direction of the remoter lymphatics. In some instances, the inflammatory process pursues its way to the trunks of the lymphatics, and even into the veins, and thus superinduces all the general consequences of an admixture of pus with the circulating current, (see Phlebitis.) This, however, appears to be of rare occurrence; Velpeau being the only observer who has met with lobular abscesses of the lungs and liver, as a consequence of inflammation of the lymphatic vessels.

When *the thoracic duct* is inflamed,[1] its membranes become thickened, and are easily torn or separated. The internal membrane has a reddened and felt-like appearance: while the external is converted along with the neighbouring cellular tissue into a dense whitish mass, which is furnished with minute vessels. The canal is filled with a fluid, at first reddish and gelatinous, but which ere long becomes firmly coagulated, and frequently assumes a purulent character. At the same time, the valves are thickened and hardened, protruding like small tumours into the interior of the distended canal, so as to impart to the internal membrane a gibbous appearance. The duct has sometimes been found partially or wholly closed, representing a cord of the thickness of a goose quill.[2] In one case of this kind, the two ends of the duct still remained open, and were found united by an arched lateral vessel. The few cases of obliteration recorded, were however found so complicated with other organic changes, that little can be deduced from them as regards the distinctive characters of the affection.

The *lymphatic glands* frequently become inflamed, either separately or in conjunction with the lymphatic vessels. The affection is both acute and chronic, and may be of a traumatic, of a rheumatic, or of a septic nature. It may occur sympathetically, from disease in neighbouring organs, or from pseudo-formations deposited within the glandular texture,[1] tubercles, for instance. In the *acute form* the lymphatic glands undergo considerable tumefaction within a brief space of time. At first, their texture is firmer than before, and of a lively red brown hue; the blood-vessels passing into the glands are gorged with blood, so that slight extravasations sometimes ensue; the neighbouring cellular texture is saturated with serous exudation, which soon coagulates, causing adhesions within the whole circumference of the gland. At this period, coloured injection has been successfully propelled from the lymphatic vessels through the inflamed organ, which, according to Gendrin, is no longer possible at a later stage. As inflammation advances, the texture becomes softened in a high degree, and assumes an ash-gray colour. Pus now begins to form, at first only at scattered points, but progressively in larger masses, until, at length, the whole gland is converted into one fluctuating abscess, which is enveloped by the indurated cellular texture, as in a capsule. In other instances, suppuration first commences in the cellular texture, and gradually spreads around the gland, until the latter becoming isolated within the sphere of suppuration, finally dissolves and wastes away.

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[1] Vide Andral and Gendrin, l. c.

[2] A. Cooper, Andral (l. c.), F. Nasse (Leichenœffnungen, p. 144), Rokitansky (Oesterr. Jahrb. N. F. (new series) vol. xvii, p. 319), Albers (Beob. vol. i, p. 61), Otto (Pathol. Anat. p. 368.)

In the *chronic form*, the cellular texture is not involved to the same extent, and its induration is confined within narrower limits. The glandular texture does not all at once pass into a complete state of softening, but collections of pus form here and there within little cavities. The whole gland, by degrees, becomes enormously enlarged; for the parts situate between the purulent collections, become saturated with plastic effusion, and swell out into lardaceous septa and bands, which now separate the different abscesses, now admit of their communicating. The pus is often mingled with other substances, as blood and fibrinous coagula. It does not readily make its escape, and when it does, not after the manner of a simple abscess; for it issues through numerous fistulous channels, which permeate the indurated and lardaceous cellular tissue of the vicinity. If the pus do not soon find an exit, it will remain pent up for years, and the swollen glandular mass will meanwhile slowly diminish in bulk, the pus becoming inspissated by the gradual absorption of its liquid parts, until it at length assumes the character of moist chalk, and is ultimately converted into a calcareous concretion. The gland then becomes partially, if not altogether, reduced to a mere capsular cicatrix.

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[1] The tubercular and carcinomatous affections of individual groups of lymphatic glands will be treated of in the progress of the work, partly as distinct forms, partly in connexion with other diseases upon which they are dependent.

Inflammation of the lymphatic vessels and glands is observed as a *sequence* of very various and dissimilar diseases, but more especially of such as either result from the introduction of noxious, poisonous, or contagious substances, or as represent some constitutional cachexia. Sömmering[1] has enumerated a long series of disorders, in all of which the lymphatic system is, in one way or another, primarily or secondarily engaged. It will here suffice to mention briefly the heads:

*Poisoned wounds,* such as those contracted during the dissection of dead bodies. Mischief here takes place by the absorption of decomposed animal matter through the wound. *The sting of insects and the bite of serpents* deserve also to be mentioned. If in these cases the affection limit itself to the lymphatic vessels, as is more frequently the case, there is far less danger than when the veins become simultaneously inflamed.

*The plague.* The contagious matter of the plague appears to act chiefly through the medium of the lymphatic system. This is rendered evident by the earlier observations cited by Sömmering,[1] and abundantly confirmed by the numerous recent researches of Bulard.[2] The latter observer found the only constant affection,—and which constituted the basis of all the others,—to be a specific inflammation of the lymphatic glands, taking its rise for the most part in the extremities, in the inguinal region and in the axillæ, advancing from thence to the groups of glands within the thorax and the abdomen, and seeming ultimately to determine a general and peculiar decomposition of the blood. The swelling of the glands often increases to the size of a goose-egg, and is conjoined with induration or softening, and finally with purulent or ichorous destruction of the glandular texture. Frequently the surrounding cellular tissue is but little implicated; occasionally, however, particularly in the abdomen and in the thorax, it becomes the seat of extensive sanguineous extravasation.

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[1] L. c. page 15.

[2] La Peste Orientale. Paris, 1839.

Notwithstanding the extreme disorganization of the lymphatic glands, Bulard found the lymphatic vessels free from all organic change.

*Typhus.* The mesenteric glands which correspond to the diseased portion of intestine are found inflamed, but in a peculiar manner in typhus; their texture is infiltrated with a yellowish-white matter; they are generally tumefied and softened; their blood-vessels are, for the most part, gorged with blood, and in a few instances even purulent formation is witnessed. This affection frequently extends to the glands beneath the pancreas, in the vicinity of the *receptaculum chyli;* but never to the lacteals and the thoracic duct, so far as I have seen.[1]

*Syphilis.* With the transition of the syphilitic virus to the lymphatic system we are most familiar. It is sometimes possible to trace the inflamed cords of the lymphatic vessels from the ulcers on the genital organs to the upper inguinal glands, which glands, together with the neighbouring cellular tissue become inflamed, and then suppurate. They are, however, not always destroyed at once by this process: for indurated fragments frequently remain, and continue, for a length of time, to occasion renewed suppuration or obstinate fistulous ulceration. Two different kinds of bubo are to be distinguished; the one resulting from the direct reception of the syphilitic virus, the other from purely inflammatory action. Ricord found by numerous trials with inoculation, that the pus of the first species is alone capable of transmitting syphilis.

*Porrigo.* When porrigo, more especially *P. favosa,* overspreads a large surface, the glands at the occiput, and from thence downwards, commonly become tumefied and painful. It is however undetermined whether this phenomenon be of a sympathetic or of a true specific character.

*Elephantiasis,* according to Allard, consists in a chronic inflammation of the lymphatics of the affected parts, and ultimately of the remainder of the body.[2]

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[1] For further details, see the second Volume.

[2] Allard, de l'inflammation des Vaisseaux absorb. lymph. Paris, 1821. Compare Sömmering, l. c. p. 102.

The lymphatic vessels in the neighbourhood of inflamed organs, of abscesses, of ulcers and of wounds, often contain *pus*, which, whether in a fluid or in a coagulated state, obstructs their canals as far as the next group of lymphatic glands,—as is manifested by pale, yellowish, knotted cords. This is most frequently observed in the lymphatic vessels of the lumbar region in puerperal phlebitis,[1] without any traces of inflammation being, in the majority of instances, discoverable in the membranes of those vessels. The question then arises: how does this pus make its way into the lymphatic vessels, since it is not generated there by an inflammatory process affecting their parietes? From the size of the globules it is not likely that it could have been taken up by absorption like other fluids;—it would rather seem to have introduced itself through the open mouths of such lymphatic vessels as lay within the range of the suppuration and have become partially destroyed[2] by it.

The same explanation may apply to *blood* said to have been found in the lymphatic vessels, and even in the thoracic duct. Actual blood-globules cannot be supposed to have entered these vessels by absorption. Either they have penetrated through injured portions of the vessels, or else it is simply the colouring matter of the blood that has become blended with the normal lymph. Occasionally general decomposition of the fluids seems to take place, imparting an abnormal taint to the contents of all the vessels.[3]

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[1] Vide figure in Carswell, fasc. viii, pl. iv, fig. 2.

[2] Compare, with reference to this disputed point, the very elaborate article of Duplay (Arch. gén. 2e. sér. vol. x, p. 308, 2 art.)

[3] A striking example of the kind is given by Breschet. (Le Syst. lymphat. sous les Rapports anat., phys. et pathol. Paris, 1836, p. 293.) A man, aged 42, had, five days before death, slightly injured himself in shaving. At the wound there arose a circumscribed swelling, with the appearance of vesicles, and a subsequent scab,—altogether remotely simulating malignant pustule, but unaccompanied with any serious constitutional disturbance. A few hours prior to his sudden and almost imperceptible dissolution, vomiting came on, but presently ceased. The cellular texture of the cheek and neck were found infiltrated with serum; the lymphatic glands of the thoracic and abdominal cavities swollen, blackish-red, as if drenched with blood, some of them exhibiting slight blackish extravasation. All the lymphatic vessels, including the thoracic duct, were gorged with a dark sanguinolent fluid. (Vide plate iii, op. cit.)

## SECT. II.—DILATATION OF THE LYMPHATIC VESSELS. PARTIAL DILATATION (HYDATIDS),—GENERAL DILATATION.

The instances are frequent in which the caliber of lymphatic vessels is preternaturally large. Nor are we able to account for this phenomenon, unless where mechanical causes have obviously prevailed, such as tumours, incarceration of parts, &c. Sömmering[1] notices numerous cases of dilated lymphatics.

Partial dilatation of these vessels, occurring between two valves, assumes the character of hydatids, as Bidloo stated, and as was proved by a case related by Meckel.[2] I have myself accidentally met with a remarkable specimen of hydatid distension of the lymphatics in the groin of a man who had died of pneumonia. Some branches of those vessels were cylindrically dilated,—several distended into small cysts,—and this cystiform dilatation extended to the lymphatics permeating several of the inguinal glands. For there were seated upon these glands spherical and oval-shaped cysts, from the size of a cherry downwards. They were furnished with a thin covering, and filled, some with a watery fluid, some with clear transparent jelly.

The most remarkable instance upon record of *general dilatation* of the lymphatic vessels is given by Breschet, as taken from Amussat.[3] A youth aged 19, had for years a swelling in both groins, for which he wore a truss. Suddenly he was seized with violent pains in the abdomen and in the tumefied parts,—great anxiety and fever—accompanied by rapid sinking of the vital powers, which ended in death. On inspection of the body the tumours were found to contain pus, and to extend far into the abdominal cavity, beneath the peritoneum. They consisted of lymphatic vessels, very greatly dilated, and so twisted and interwoven amongst each other as to resemble the seminal vesicles. The whole of the lymphatic system, up to the point of inosculation with the left subclavian vein, was dilated,—and, what is particularly notable,—the inguinal glands had altogether disappeared, having been resolved into the above-mentioned lymphatic enlargements.

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[1] In his often cited work De Morb. Vas. Absorb.

[2] Path. Anat. vol. ii, part i, page 260.

[3] Loc. cit. p. 260, plate iv. The same case is figured by Carswell, fasc. ix, plate iv, fig. 4.

Dilatation of the *receptaculum chyli* has been observed by Rokitansky,[1] and by Albers,[2]—of the *thoracic duct* by Andral,[3] by Baillie,[4] by Otto,[5] and twice by myself. These several cases are, however, too unconnected, too imperfectly described, and too much complicated with other diseases to allow the deduction of any general inferences.

## CHAPTER II.

### DISEASES OF THE VEINS.

### SECT. I.—PHLEBITIS. VENOUS INFLAMMATION.

No subject more amply illustrates the essential services which the science and art of medicine have derived from pathological anatomy than that of phlebitis. By this study many a dark point in the phenomena of disease has been either thoroughly elucidated, or at all events, rendered more comprehensible. We need only refer to the so termed malignant intermittents consequent upon wounds and surgical operations, — to certain typhoid conditions, puerperal diseases and the like. John Hunter, the elder Meckel, and Peter Frank, were the first to commence the investigation.[6] Since then inflammation of the veins, as well as of the arteries and lymphatics, has been observed under every variety of circumstances. By many, indeed, too wide a field has been awarded to the inflammation of vessels;—thus numerous errors have been disseminated. This has, however, only caused the subject to be the more minutely studied,—to have had assigned to it characters more determinate, and limits more within the compass of practical experience.

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[1] Oesterr. Beob., vol. xvii, p. 319.

[2] Beob. vol. i, p. 60.

[3] Précis d'Anat. path.

[4] Anat. des krankhaften Baues, p. 57.

[5] Path. Anat., paragraph 212, note 2.

[6] Balling (Zur Venenentzundung—Wurzburg, 1829), in his first volume, gives a very good and detailed history of phlebitis up to 1829.

The veins, owing probably to their double function as vessels of return and of absorption, as also to the protracted sojourn of their contents at any one point, are more prone to inflammation than any other system of vessels.[1] They may participate in the inflammation of organs or parts of the body which they traverse, or become inflamed by irritant substances coming in immediate contact with their internal or external surface. It has been asserted by Gendrin and others, that reaction more readily manifests itself in the external than in the internal membrane,—the latter being but slightly susceptible of irritation, and suffering, both least and latest, any inflammatory change. This assertion, however, does not appear to me to be altogether borne out by facts. For we frequently observe that substances, not in themselves irritant, as, for instance, coagulated blood, pus, &c., are sufficient to induce internal inflammation; nay, examples occur in which a very slight alteration in the qualities of the blood will produce this effect. The internal membrane of the veins reacts, indeed, upon the application of irritant substances, almost as quickly and intensely as the serous membranes. In this reaction, doubtless, the vascular substratum plays the principal part: the lining membrane yielding merely to the alternations of endosmose and exosmose, and not suffering any organic change until a later period. In this respect it will appear not unworthy of notice, that those portions of the venous system which are composed exclusively of the internal membrane of the veins, with a very scanty provision of surrounding cellular tissue—like the corpora spongiosa—are very rarely, and never extensively, the seat of true inflammation.[2]

The doctrine of phlebitis is naturally divisible into two heads: the one restricted to the consideration of the purely local, primary phenomena, the other to the general, secondary consequences, diffused throughout the whole system. The latter, first placed in their true light by Dance and Arnott,[1] form one of the most interesting and important subjects of pathology. We first proceed to consider the former.

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[1] The lymphatic vessels are similarly circumstanced, but with this difference, that, possessing a lower grade of vitality, they are less susceptible of irritation. Their simple structure, compared with that of veins, explains their slight tendency to disease in general, and to inflammation in particular. It is quite otherwise with the lymphatic glands. (Refer to the foregoing chapter.)

[2] The occurrence of inflammation in the corpora cavernosa has been doubted. Arnott has, however, quoted an example from the Ephém. méd. de Montpellier, vol. i, p. 126; and another is given by Bichet (Arch. gén., Juill. 1841, p. 313.)

When the inflammatory process develops itself in a vein, its coats, internal as well as external, become at once reddened. The redness is, at first, observable throughout the whole extent of the inflamed portion; and a gradual transition to the natural colour discernible only towards its confines. So soon, however, as the disease gains ground, the parts become irregularly spotted, marbled, occasionally streaked, and at length display every variety of shade, from the natural colour to a dirty violet on the one hand,—to a deep scarlet on the other. This discoloration does not depend upon the development of minute vessels upon the internal membrane,—for on the most careful inspection I have never been able to detect any,—and in no respect differs from the redness of imbibition, except in the mottled alternation of its various shades. Again, the cellular texture surrounding the vein exhibits an incipient infiltration of a faint-red serous fluid, together with a dense network of delicate little vessels, which, in the larger veins, are distinctly seen to extend to the cellular coat of the vessel. A double consequence now appears to ensue,—and in most instances very rapidly: namely, deposition of an inflammatory product within the canal of the vein, and a change in the blood itself. For, as the morbid blood vitiates the membranes of the vein, producing inflammation, so, in like manner, these membranes when once inflamed, exercise a reflex action upon the blood; the first sign of which is, probably, the inflammatory redness of imbibition already described. The further changes consist in the formation of fibrinous deposit, at first loosely connected with the internal membrane of the vein by means of a tenacious mucus-like substance, but which subsequently adheres more and more firmly through the medium, as it were, of cellular tissue.

Should any one imagine this false membrane to be not the product of plastic effusion from the coats of the vein, but an immediate deposition from the blood—let him refer to the experiment of Gendrin.[1] Having secured a portion of an artery between two ligatures, and entirely cleansed it of blood, that experimentalist discovered, after throwing in an irritant injection, a plastic substance deposited within the part so insulated, filling up the whole caliber of the vessel; and he affirms that similar trials with veins led to the same results. Hence it is natural to infer that in vessels containing blood the plastic product partly exudes from the parietes of the vessels, and is partly deposited from the blood.

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[1] Dance (Arch. gén. de Méd., Déc. 1828, Janv. et Févr. 1829); Arnott, (Medico-chirurg. Transactions, vol. xv, Lond. 1829.)

Fresh fibrinous layers are now continually being deposited, and in a proportionately very short time a plug is formed which fills the entire caliber of the vein. This plug is of a pale brownish or yellowish-gray colour, and is made up of concentric layers, of which the central portion is usually softer than the rest. The nucleus is, however, now and then somewhat more solid than the layers immediately surrounding it, and consists of very firmly coagulated blood of a dark, almost of a black colour. So soon as a fibrinous plug of this description is established, the red and violet speckled colouring abates in intensity, and the internal membrane, losing its smoothness and polish, assumes a dull, velvety or slightly puckered appearance. The external membrane appears thickened, turgid, and soon becomes adherent to the cellular tissue, which, in its turn, has been rendered firmer and paler from the effusion of plastic lymph. Both membranes are still readily distinguishable, and even separable, from each other; the consistency of their texture is, however, impaired, and they are easily torn. In this state of things a vein, when cut asunder, does not collapse, even after the plug has been removed, but, on the contrary, its caliber remains open like that of an artery. This is more than ever the case when the surrounding cellular texture has acquired firmness by the condensation of the inflammatory product effused into it, or when it puts on a brawn-like character and intimately coalesces with the external membrane of the vessel. This point is important, for we shall have hereafter occasion to show, that in dilatation of the veins a similarly disorganized condition of the cellular tissue, and a consecutive permanent distension of the venous canals frequently occur, and, under certain circumstances, even extend to the minute venous twigs. When suppuration follows the excision of hemorrhoidal and other varicose tumours, the risk of pus getting into the circulation, with all its concomitant perils, will be proportionate to the number of open vessels within the wound. The fibrinous coagulum described as plugging up the canal of the vein, extends, both above and below, far beyond the limits of the portion originally inflamed,—the plug, however, ceasing by degrees to be made up of concentric layers, gradually passes into an attenuated coagulum, more or less tinged with the colour of the blood, and little, if at all, adherent to the walls of the vessel. In all the branches of veins which immediately lead to the plugged portion, save those which have other channels by anastomosis, the blood stagnates and coagulates, and, the larger the trunk of the inflamed veins, the farther back of course will this effect be observable. The plug is shorter above, and seldom extends beyond the point of junction with the nearest venous trunk.

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[1] Loc. cit.

The inflammation has, however, a great tendency to spread farther and farther, and although most frequently in the direction of the heart, it may likewise take an opposite course, particularly in cases dependent upon a morbid state of the blood itself. Its advance is not, in most instances, continuous, but interrupted at intervals with healthy vein, of which sound portions are, by means of the numerous anastomoses, kept open to the circulation.

When the inflammatory process is arrested or exhausts itself at this, the adhesive stage, all those changes take place which we shall afterwards have to describe as modes of termination. This is rare, however, for it is a characteristic of phlebitis,—which renders it of more serious import than the inflammation of other systems of vessels,—that in the great majority of instances it leads inevitably to suppuration. Herein alone lies the great risk in so many cases of apparently trifling operation, such as the tying of varicose veins, &c.

To determine the mode in which pus is formed within inflamed veins, is one of the most weighty points connected with pathology. The subject indeed, strictly speaking, belongs to *general* pathological anatomy. Nevertheless, its importance is sufficient to warrant a brief account, in this place, of the present state of our knowledge respecting it. The principal question is: whether the pus found in the veins be the result of secretion from the inflamed surfaces, or of direct metamorphosis of the blood itself. Gendrin believed that he had observed, by help of the microscope, a direct change of the blood-globules into pus-globules, and he endeavoured to prove this by the following experiment: having, by means of a double ligature, isolated a portion of an artery or of a vein, he caused it to inflame by injecting an irritant fluid. He then readmitted the current of blood, and afterwards confined it by definitively drawing the ligatures together again. Hereupon suppuration commenced in the vessel, and the blood becoming first coagulated, and then deprived of its colour, was by degrees altogether converted into pus. This is the experiment so frequently cited, and by many held to afford incontrovertible evidence. More recently M. Donné has employed the microscope for the purpose of demonstrating the conversion of blood into pus. Having mingled the two substances in the proportion of 8 to 1, he traced all the gradual changes wrought in the blood-corpuscles, until, after the lapse of twenty-four hours, none but pus-globules were discernible. On the other hand, Gluge (as formerly Vogel, in opposition to Gendrin) has shown, that in water, and in every other kind of liquid capable of dissolving their capsules, the blood-corpuscles undergo precisely the same modifications of form as those described by Donné. Hence it may be reasonably inferred that the blood-corpuscles become destroyed, and that ultimately pus-globules alone are to be met with in the fluid serving for the experiment,—not that the individual blood-corpuscles are transformed into pus-globules. Gluge could not detect any alteration in the blood, in consequence of inflammation, beyond the formation of what, both by himself and by Valentin, were termed "composite inflammation- and exudation-globules." It would make a material difference could it be shown that the liquid resulting from the solution of the blood-corpuscles were, at least partially, capable of conversion into organic elementary cells, which, in consequence of the existing inflammation, assumed the form of pus-globules. This view is, however, merely hypothetical, for I have not as yet been able to subject it to the test of experiment. Some light may, perhaps, be thrown upon the point at issue, by the further prosecution of E. H. Weber's interesting inquiries concerning the minute globules that slowly revolve along the parietes of the vessels, and which, according to that excellent observer, are blood-corpuscles modified through the process of nutrition.

The assumption that the pus is secreted by the coats of the veins is founded upon analogy. Vogel demonstrated the transition of epithelium-cells into pus-globules, and the fact has since been amply confirmed by Henle, who, availing himself of the discovery of Schwann, "that all organic bodies are developed out of nucleated cells," showed, partly by direct observations, partly by analogical reasoning, that out of these "primary cells" forms may spring, either normal or pathological, as the case may be. Thus, pus-globules would originate as the product of inflammation. In accordance with these views, the puriform masses generated within the veins would be developed as follows. First of all the cells of the epithelium-lining, discovered by Henle, separate from the internal membrane of the vein, so as to give to the inner surface of the vessel the dull appearance already described, and to render it more susceptible of a morbid tinge from imbibition. The next change affects the passing blood-corpuscles, which assume a spheroid, or else a gibbous appearance, advance with a slow revolving movement, or cling to one another, parting with their serum (plasma, according to Schultz), and with their pigment. The internal membrane of the vessel generates new, imperfect epithelium-cells, which mingle with the altered blood, and finally actual pus-globules, which, when congregated in sufficient number, completely arrest the current of the blood, and affect the blood-corpuscles in the manner already pointed out. The simultaneous effusion of both fibrin and albumen[1] now serves to complete the formation of a plug which differs in external character according to its more or less rapid development, and to the varying proportions of its constituent parts. The plug thus originating afterwards undergoes further changes. It ought not, however, to be concealed that this description rests, for the most part, upon analogy only, the test of microscopic investigation having, as yet, demonstrated the above processes only in the smallest vessels, and by no means in the larger ones.

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[1] Gulliver (Lond. Med. Gaz., March 1839) maintains that the puriform fluid in veins contains no pus-globules, but consists merely of liquefied fibrin. Concrete fibrin, kept in water at the temperature of the blood, became converted, in a couple of days, into a puriform mass, which, under the microscope, displayed a multitude of globules, varying in size and shape, but not a single pus-globule. This experiment I have myself repeated with a similar result. The fibrin is transformed into an amorphous granular mass, strongly resembling inflammatory exudation. I have, however, likewise found vast numbers of genuine pus-globules in inflamed veins.

Passing from the above digression to the immediate description of the visible processes, we have first to examine what is the condition of the plug before described, when suppurative phlebitis (the hyperphlogosis of Lobstein) sets in. It is now found to be softer, especially towards its middle; it assumes a grayish, yellowish white, dotted appearance, and finally exhibits a straw colour, and a semi-fluid consistency. Its laminated structure becomes more and more indistinct, and it is finally resolved into pus, which is usually confined within a fibrinous layer more or less thin, and rarely found loose within the vein. But the contents of the veins being unceasingly propelled towards the heart, the more or less solid products of inflammation are necessarily conveyed beyond the original site of inflammation. For this reason it would be premature, were we at once to conclude, in examining a body, that the part of a vein at which we might happen to find a pus coagulum must be the true seat of the disease. That seat is frequently remote, and difficult to discover; thus betwixt a purulent coagulum in the inferior vena cava, and a gangrenous spot or a varicose ulcer on the leg, the whole extent of the iliac and crural veins, together with their deep-seated branches, shall be found perfectly healthy, whilst one or more branches of the saphena vein alone bear all the evidence of intense inflammation.

But since pus by mingling with the blood causes its coagulation, a decided hinderance is thereby offered, in the majority of instances, to the product of inflammation passing along with the venous current. The pus becomes isolated by the coagulation of blood, both above and below the place of its formation, and is thus cut off from the remainder of the blood. Cruveilhier terms this the *sequestration* of veins. Under such circumstances, the pus may be gradually removed by the process of absorption, the vein in the meantime becoming obliterated; or it may make for itself an outlet through the parietes of the vein. Then abscesses, varying in size and number, according to the amount of inflammation, form beneath the skin, or between the muscles, and the patient is thus protected against the dangerous consequences of a general infection of the circulating fluid.

The formation of matter being brought about in the manner above related, within the inflamed vein, its membranes have likewise to undergo a further change. Their colour now inclines to a grayish white; they become softened and thickened; are no longer to be distinguished from one another; and form, in conjunction with the surrounding textures, a nearly uniform membranous layer, of a lardaceous aspect and character. By and by, a turbid, puriform fluid is often found deposited at intervals in the cellular tissue; in some instances, where the suppuration is vigorous in the vicinity of the vein, the latter traverses the purulent channel for a considerable space, denuded in its entire circumference. Here the membranes of the vein gradually soften, and at length melt down, so to speak, until no further vestige of their texture is discernible within the common centre of suppuration.

When the above venous *sequestration* does not take place, or but imperfectly, and the pus or the softened fibrin passes at once into the general circulation, all those phenomena ensue, which, assuming first the type of an irregular intermittent, and subsequently a typhoid or putrid character, appeared so enigmatical to our forefathers.

The organic changes which are to be viewed as results of the morbid condition of the blood, display themselves in every variety of organ, throughout the whole body. They are all referrible to stagnation of the blood, and are divisible into such as occasion a stagnation and interruption of the sanguineous current in the central portions of the vascular system, and into such as have their seat in the capillary system.

The first series of changes consist, according to the observations hitherto made, in the formation of pus and in coagulation of blood within the large venous or arterial trunks—even in the heart itself. It would appear that, in such cases, the product of inflammation, be it pus or finely-divided fibrin, follows the course of the blood towards the heart, but advancing more slowly than the uncontaminated blood, accumulates, invests itself again and again with fresh layers of coagulum, and ends by entirely closing up the caliber of individual vascular trunks. This occurs most readily in parts through which a large quantity of blood has to pass within a brief space of time, and in which, therefore, the pus-globules and the fibrinous particles accumulate the more rapidly.

This coagulation of the blood, consequent upon phlebitis, has been observed most frequently in the pulmonary artery and its branches. The plug which usually forms here, perfectly resembles that of the adhesive inflammation of vessels. It is of a pale brown colour, with here and there a yellowish spot, is composed of concentric layers, and attached more or less firmly to the parietes of the vessel. These concrete masses spread to the minutest extremities of the pulmonary artery, in most instances of one side only, and death presently ensues, so soon as the main trunk gets choked with them. Coagulation of the blood in the pulmonary veins has been observed in puerperal phlebitis, by R. Lee,[1] and by myself, in phlebitis consequent upon uterine carcinoma.

In other rarer cases, similar coagulation occurs in the right cavities of the heart. Extensive *polypi cordis,* as they are commonly termed, are then found, of a grayish or pale violet colour, and displaying, more or less distinctly, a stratiform and fibrinous structure; internally they are sometimes found considerably softened, and occasionally even containing liquid pus. Externally they intertwine in various ways with the columnæ carneæ, and with the valves of the heart, being overspread with congealed black and gray spotted blood, or marbled with purulent streaks.[2]

To produce the coagulation above described, it would appear indispensable, not only that a certain amount of morbid matter should pass into the circulating mass, but likewise that there should exist a peculiar predisposition of the body generally.

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[1] Med. Chir. Transact. vol. xix.

[2] Many of the cases related by Bouillaud as examples of endocarditis are of the above character. The puriform fluid in the coagula of the heart's cavities does not appear always to be true pus. In two instances in which I examined it under the microscope, it proved itself to be an amorphous, granular mass, of the nature of dissolved fibrin.

For, if the observation of Gluge be not incorrect, pus-globules are found in the blood contained within the heart in phthisis; and yet little or no coagulation takes place,—owing probably to the globules only passing one by one, and in slow succession, into the blood.

A most important sequel of phlebitis is that which involves secondary organic changes within the capillary system. These have been designated by the appellations of "*lobular inflammations*" and "*lobular abscesses.*" They most frequently occur in organs through which the greatest portion of the blood is propelled within a short space of time; viz., in the lungs and liver. They are rarely observed in the spleen, in the kidneys, in the external skin, and in cellular tissue,—more rarely still in the brain, in the eye, and in the muscles. Serous membranes are little prone to this puriform effusion—the synovial membranes and the pleura rather more so than the others.[1] Puerperal peritonitis is an exception; it is, however, frequently the result of concurrent metritis. Veins distant from those originally diseased likewise appear to be obnoxious to this secondary suppuration.

The peculiar character of these abscesses in the most dissimilar organs (of which a more minute description will be found in the chapter on Pneumonia), has long attracted the attention of medical men, to whom they appeared particularly puzzling, when originating from external mischief. The first observed were hepatic abscesses, following injuries of the head; and, under the impression that the liver was the only organ liable to become affected in the manner and under the circumstances in question, the most untenable hypotheses were urged by way of explanation. Dance and Arnott however established, from an impartial scrutiny of coincident facts, that abscesses arise in many other organs besides the liver, from injuries both of the head[2] and of other parts. They further proved, that such abscesses exhibited the same character and became developed under similar symptoms with those collections of pus, the connexion of which with phlebitis they had before clearly demonstrated.

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[1] Besides the treatises of Dance and Arnott, compare Velpeau (Rev. Méd. 1826 and 1827, Arch. gén., vol. xiii), Balling. (l. c.), Th. Helm (Oesterr. Jahrb. vol. xxiii, p. 1), Schuh. (ibidem, vol. xxv, p. 3.)

[2] The above-named pathologists were the first to draw correct inferences from these facts, which were previously known to Morgagni and others.

On such convincing evidence, we are bound to ascribe the phenomena in question to a phlebitis in which the pus formed is not isolated, but mingles with the general sanguineous mass. Whilst, however, in many instances of external injury,—fractures of the skull, amputation, &c.,—minute anatomical investigation has confirmed the assumption of actual phlebitis; still there are cases in which distinct venous inflammation cannot be detected in the dead body. But, even then, the course and the symptoms of the disease, as well as the appearances after death, furnish us with ample evidence of contamination of the blood with pus; we must, therefore, take it for granted, that either the phlebitis had escaped discovery, owing to the minuteness of the inflamed vessels, or else that pus had at once passed by absorption into the blood, without any previous phlebitis. The former case may indeed be often assumed, for it is extremely difficult after the operation of lithotomy, for instance, to single out amid the thickened, lardaceous and partially ulcerated cellular tissue, the smaller veins of the vesical plexus, or to examine accurately all the venous sinuses of the womb, in the case of a female dying in child-bed. Still the other supposition cannot be regarded as wholly untenable, although the absorption of unaltered pus through the capillaries is hardly admissible, upon physical grounds.

Another question still presents itself, namely, whether the pus formed within veins at the part originally inflamed, be substantively transmitted through the medium of the circulating current to the lungs, the liver, &c., to accumulate at certain points within the latter; or whether it be actually generated in the parenchyma of those organs. The former opinion was at one time zealously maintained, and numerous observations were adduced in evidence of such metastasis. More recently, however, the latter view has prevailed; and although, in these processes, there is still much that remains to be cleared up, yet an unbiassed comparison of the facts has furnished an explanation adequate to the majority of cases.[1]

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[1] The subject is treated of in detail in Vogel on Pus, &c., pp. 200-19.

With regard to lobular abscesses, there cannot at the present day be any further question of the pus being conveyed to them, *exclusively* and *in quantity*, by the circulation. The best authorities have repeatedly asserted that these collections are not at once purulent at the outset, but that knots form, of from the bigness of a pea to that of a walnut, become infiltrated with firm coagulated blood, and eventually suppurate. I have had opportunities of convincing myself of the correctness of this fact, with reference to the lungs, the liver, and the spleen. It may be, therefore, concluded that, owing to some obstacle, the blood stagnates at certain points, producing suppurative inflammation of the surrounding tissues.

The experiments of Leuret, Trousseau, and others,[1] and of Cruveilhier,[2] afford an insight into the cause of such stagnation; for, when putrid and other substances are injected into the veins, organic changes, perfectly analogous to those above described, are developed with the accompaniment of low typhoid fever.

The experiments of Günther[3] are the most striking of all in their results. Having injected pus into the veins of horses, he very shortly afterwards found fully-formed lobular abscesses in the lungs.

From these data we may, with some degree of certainty, infer that the pus is conveyed in substance by the veins to the heart, and forwarded from thence; but that those pus-globules which have reached the capillaries of the lungs in their entire state, are unable, from their size, to permeate the latter. These globules now become a central point of stagnation, (and finally of extravasation,) in the adjunct branches of the pulmonary artery, and thus determine, eventually, local inflammation and suppuration.

In this manner, phlebitic abscesses in the *lungs* are satisfactorily accounted for, as are also those which occur in the *liver*, in consequence of inflammation within the tract of the *portal system*. The origin, however, of purulent collections in other organs still remains obscure. Here, indeed, the above explanation is inapplicable, founded as it is upon the inability of the pus-globules to permeate the minute capillary vessels of the lungs. Günther found that these deposits were formed *subsequently* to those in the lungs, and believed that they originated from pus being taken up from the diseased parts of the lungs by the pulmonary veins, and thus carried into the greater circulation. Were this explanation correct, phlebitic abscesses must necessarily exist in the lungs, wherever such deposits are found in the capillary system of the greater circulation. To ascertain this, I have compared a large number of cases, observed, partly by Balling, Dance, Arnott, and others,—partly by myself.[1] Amongst them, however, there are only two,—one related by Sasse,[2] of purulent deposits in the liver, and one by Dance, of purulent exudation within the right wrist-joint, in which the non-existence of pulmonary abscesses is established by careful examination after death. Four of Arnott's cases, the first, third, seventh, and ninth, would certainly appear to belong to the same class; they are, however, not related sufficiently in detail to admit of any decided inference being drawn. It is singular, indeed, that the seat of the purulent secretion, in these four instances, was within serous sacs—in three of them, within those of different articulations, and in one, within that of the pleura. It may, upon the whole, therefore, be assumed that in some cases the substances commingling with the blood, pass through the capillary system of the lungs, without inducing any change in the pulmonary parenchyma. Vogel, without indeed assigning any reason, considers it not impossible for single pus-globules to pass through the capillaries of the lungs. It is perfectly intelligible, at all events, that the nuclei of ruptured pus-globules may pursue their course without hinderance into the greater circulation. This applies equally to fibrin altered by the inflammatory process; finely divided particles of which will, if hurried along by the circulating current, be in many cases productive of exactly the same effects as pus.

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[1] Arch. gén., vol. xi, p. 373.

[2] Nouv. Bibl. Méd., vol. iv.

[3] Rust's Magazine, vol. xi, fasc. ii, 1834.

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[1] To avoid error, only those cases have been made use of in which the after-death appearances are minutely detailed; for which reason the thirty-three cases collected by Arnott, of internal abscess resulting from external injury, could not be taken into account.

[2] Sasse (l. c.), in his report of the after-death appearances in one who had died of uterine phlebitis, says, in speaking of the liver—"its colour was of a grayish yellow, its substance soft to the touch, and equally so under the scalpel. On pressing a portion removed by the knife, a small quantity of puriform fluid, of a greenish yellow colour, escaped from the minute cavities of the parenchyma;" and of the lungs—"they had, both externally and internally, the appearance of being healthy; the bronchi and trachea were clogged with a small quantity of green frothy mucus."

It has been before stated that, either from isolation of the pus generated within veins, or from the inflammation not exceeding the limits of the adhesive process, general contamination of the blood, together with its perilous consequences, is not in all instances realized. Nevertheless a permanent, or at least a temporary obliteration of the inflamed portion of the vein will be the natural result. It is surprising in how short a space of time a plastic plug extending through numerous branches, and even filling up a tolerably large trunk, will disappear, so that canals previously impervious are again open to the circulation.[1] The dispersion of the plastic mass is probably effected not so much by absorption through the medium of the vasa vasorum, as by its positive resolution and liquefaction in the sanguineous mass itself. It is indeed matter of astonishment how those very substances which excite the most violent symptoms, when conveyed during and through the instrumentality of the inflammatory process, now become blended with the circulation without occasioning any mischief whatever. At this period, however, prudence and caution are still needful; for instances are not wanting of errors in diet, taking cold,—in short, whatever at this stage of incomplete recovery has tended either to rekindle the local inflammation or to excite the general circulation,—leading to a fatal issue.

When the inflammation has been so violent as to occasion more decided changes of structure in the coats of vessels, the usual result is a complete obliteration of the diseased vein, through which the circulation has been impeded long enough to admit of the anastomoses being amply developed. Then the activity of the vasa vasorum becomes conspicuous; a considerable number of these vessels having formed in the venous membranes, which are by this time reduced to a homogeneous, lardaceous tissue. By their agency the product of inflammation obstructing the vein is very gradually assimilated and absorbed. Meanwhile the walls of the vessel become more and more thickened, contracting in folds or wrinkles upon the narrowing canal, until at length the whole vessel is converted into a thin cellulo-fibrous cord. In relation to this point the perfectly analogous process by which the obliteration of fœtal vessels is brought about, is instructive; the only difference being this, that in the fœtal vessels obliteration is the result of a physiological process, there being simply a slender plug of coagulated blood to surmount, instead of the more heterogeneous products of inflammation. We shall see, farther on, that, under certain circumstances, even the above physiological process may become exalted into a pathological one, and attain the perilous grade of confirmed phlebitis.

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[1] This fortunate termination of phlebitis I have experienced in my own person. The whole system of the saphena, up to its insertion into the crural vein, had become blocked up by plastic lymph; even the minute twigs of the corium had, by the formation of pustules beneath the epidermis, given proof of active participation in the disease, and yet, within five weeks of the inflammation subsiding, the circulation was fully restored in almost all the veins, as before.

It sometimes happens that the obliteration of the canal of the inflamed vein is incomplete. Part of the plastic substance filling up the caliber has become organized, and opens a small channel for the passage of the blood. I have had an opportunity of observing this phenomenon in one instance only; here the deep-seated and superficial femoral veins were filled up with plastic, organizable matter. The coats of the vessels were completely thickened, presenting an appearance of turgidness like that which results from maceration, so as to render it impossible to recognize their original structure, or to distinguish between the different membranes. At the same time they had so intimately connected themselves with the surrounding, and in like manner thickened, cellular tissue, that they could no longer be separated from it by dissection. Nor was it easy to ascertain where the internal membrane of the vein ended and the inflammatory plug began, for firm cohesion had taken place between them. Both together formed, in the larger branches, a layer, of the depth of two Parisian lines, which towards the interior was rather soft and dark coloured, as if drenched with blood, whilst towards the exterior it exhibited a dirty white hue, and was tolerably consistent. Within these apparently solid cylinders, and running lengthwise, parallel to their axes, were one or two small venous twigs, partly enveloped already with a thin, smooth membrane. Carswell has given a perfectly similar case.[1] He is of opinion that the circulating current, in its effort to re-establish itself, dissolves, and carries away the softer central layer of the plug, so as to open the new venous passages, whilst the comparatively firm external layers gradually become organized. This explanation appears to be the correct one. The whole phenomenon, however, which from its minuteness may easily be overlooked, affords another striking example of that great power of reproduction, so variously and vigorously displayed in the vascular system. In another similar instance, I found the crural vein transformed into a whitish cord, and replete with an organized mass of the firmness and consistence of bacon fat. Betwixt this plug and the thickened coats of the vessel, round about the periphery of the former, were several little canals, which, running along the whole extent of the vein, had already begun to re-establish the circulation. In all probability the impulse of the blood from below had, during the first period of the inflammation, here and there severed the plastic plug from the parietes of the vessel;[2] subsequently, the above plug becoming organized, and the intervening blood absorbed, several peripheral channels would form, instead of a single central one.

It will be seen from what has been premised, that phlebitis may originate under a great variety of circumstances. It is as frequently the consequence of slight external lesions, venesection for instance, as of great surgical operations and of extensive sores. Upon the whole we may regard with alarm every instance of profuse suppuration occurring in the proximity of such veins as remain open-mouthed when wounded, either owing to anatomical situation, as in the instances of the veins of the diploë, —of the axillary veins,—of those within the uterus,—within the liver, &c.; or to some morbid change of structure consequent upon inflammation, varicose distension, and the like; or, lastly, to the surrounding cellular tissue thickening, assuming a lardaceous character, and thereby keeping the parietes of the veins upon the stretch. This is the reason why phlebitis is so frequent, and so fraught with danger after wounds of the head, and after the operation of lithotomy; and also why phlebitis artificially induced, for the purpose of obliterating varicose distensions, so readily spreads to an alarming extent, when once it gets beyond the adhesive stage.

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[1] See the well-executed figures 6 and 7, fasc. xi, pl. ii.

[2] As repeatedly seen by Cruveilhier, livr. xi.

Phlebitis attacks the small branches of veins in the proximity of ulcers, especially of the cutis, in erysipelas, in diffuse phlegmonous suppuration in tubercular cavities, &c., partially, and to an extent altogether limited. Under these circumstances, the vessels in the vicinity of parenchymatous ulcers usually become obliterated, and others are progressively developed, in their stead, out of the capillary system. This is often exemplified in a very remarkable manner in tubercular phthisis. It may however be asked, whether this obliteration may not be independent of inflammation, and the effect only of augmented vascular activity not exceeding certain defined limits, as in the case of the umbilical artery and vein; or whether the degree of inflammation, by which the exulceration of the part is accomplished, be communicated to the neighbouring veins; or, lastly, whether pus actually penetrates into those veins and thus occasions their closure.

One of the most perilous forms of phlebitis is that which arises from the absorption of *septic* matter, for instance, through scratches or wounds incurred during the dissection of dead bodies undergoing decomposition. A general contamination of the fluids within the body itself may, under particular circumstances, occasion venous inflammation. I have witnessed it in the veins of both the inferior extremities of a woman who, for years, had been suffering from a general syphylitic taint, and had latterly become phthisical. A similar case is related in the 'Rapport de la Société Anatomique' for August 1834. In both cases there was considerable œdema of the lower extremities; in that of the woman, who was under treatment at the Leipsic hospital, the course of the disease was precisely similar to that of *phlegmasia dolens.*

It is hardly possible to decide whether, in puerperal diseases,[1] septical influences co-operate, or whether the same causes alone prevail as in simple wounds and injuries, with a disturbed and imperfect process of suppuration. At all events, *puerperal phlebitis* is one of the most frequent varieties. It develops itself with uncommon rapidity whenever, after expulsion of the fœtus and of the placenta, the uterus does not contract properly, so that an extensive raw surface with open-mouthed veins is exposed.[1] In such cases the internal, spermatic, and a large portion of the branches of the hypogastric veins, sometimes of both sides, but more commonly of one side only, exhibits various stages of inflammation; and, whenever the inflamed parts are not partitioned from the great trunks by means of the adhesive process, all the consequences before described of general infection of the blood, ensue. The venous sinuses in the substance of the uterus are distinctly distended with pus, sometimes fluid, sometimes as if coagulated, and then adhering more or less firmly to the parietes of the vessels, winding through every sinuosity and ramification, and, when removed, readily liquefying or yielding to pressure. In many cases these pus-conduits, appearing like little abscesses, are exposed by every incision into the uterus; frequently, however, more careful examination is necessary for detecting the source of suppuration. That nothing may be overlooked, it is necessary to devote particular attention not only to the locality where the placenta had been attached, but likewise to the convolutions of veins which lead towards the cervix uteri. The branches of the internal spermatic and of the hypogastric veins usually contain grumous, soft, coagulated blood, speckled with grayish and yellowish dots,—or a firm plug consisting of concentric layers, or else more or less fluid pus.[2] The substance of the uterus, according to the degree and duration of the affection, is either slightly infiltrated with serum about the venous sinuses only, and otherwise healthy, or it is inflamed and softened, or in a state of putrescence. In more extensive disease we find the ovaries inflamed, with abscesses in their interior. The lymphatics are frequently involved, and filled with pus. In most instances there is concomitant peritonitis in various grades of intensity.

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[1] R. Lee, M.D. (Researches on the Pathology, &c., of the Diseases of Women); Th. Helm (über Puerperal-Krankheiten, 1839); Kiwisch (die Krankheiten der Wochnerinnen, 1840).

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[1] Dance showed that fluids, injected into the vena cava inferior, penetrated with perfect ease into the uterine cavity, through that portion to which the placenta had adhered.

[2] Compare figure in Cruveilhier, livr. iv, pl. vi (copied in Froriep's Klinische Kupfertafeln [clinical plates], plate xxvi), and livr. xiii, pl. i, ii, iii.

Sometimes the inflammation, without distinct manifestation in the uterus, spreads from the hypogastric to the iliac and crural veins, and to their different branches,—more frequently of the left than of the right extremity. Thus is developed *phlegmasia dolens*, which is the more completely characterized when not only the saphena but likewise the deep-seated veins are inflamed and obstructed. For, if one or the other of the larger trunks, or even an extensive anastomosis remain open, or if suppuration rapidly supervene, the circulation is only partially or temporarily impeded, and of course neither the œdema nor the tumefaction is so marked as in what is commonly described as phlegmasia dolens. Although many high authorities agree in regarding the disease in question as essentially phlebitis,—although it has been observed as the result of phlebitis in females not in child-bed,[1] in fact even in males,—some men still persist in ascribing the affection either to an inflammation of the lymphatics, which in many instances certainly does coexist, or of the cellular tissue, or of the nerves of the thigh and leg; or again, to disease of the ligamenta uteri rotunda, or to milk-metastasis. Cases may be cited in support of each of these opinions; the subject accordingly demands further investigation. It would be improper to decide in any case until after a searching examination of the veins of the affected limb or limbs, and not of these alone, but likewise of the iliac and inferior cava. Graves and Stokes[2] mention an instance in which, although there was inflammation of the saphena of both inferior extremities, one only of the latter presented features resembling those of phlegmasia dolens. In such instances we have duly to consider all that has already been advanced with reference to the character of the inflammation, and to the degree and extent to which the anastomoses and the various trunks are involved: for phlebitis in the lower extremities does not necessarily lead to phlegmasia dolens.[3]

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[1] As in the case before adduced by myself, or in the uterine carcinoma, of which Cruveilhier (livr. xxvii) narrates several examples, giving, at the same time, a very beautiful figure of inflammation of the crural veins (plate iv.)

[2] Dublin Hospital Reports, vol. v.

[3] Graves and Stokes, moreover, state that, in phlegmasia dolens the swelling

In *gangrene of the extremities* the trunks of the veins have almost invariably been found obliterated, particularly in *spontaneous gangrene;* an opinion has therefore been promulged, that it depended upon inflammation of the veins in question. An abiding obstruction to the circulation within a part which is connected with the rest of the body at one point only, must undoubtedly lead to gangrene, as is indeed proved by numerous examples of its being brought on by adhesive inflammation invading simultaneously the trunks and ramifications of an artery. In such instances the cause that gives rise to the arteritis might at the same time occasion inflammation of the corresponding veins, without the phlebitis, although *preceding,* being of necessity the efficient cause of the gangrene. For it might be generally shown to be the gangrene that determines the inflammation of the proximate veins, through the irritant action of the gangrenous ichor. In the arteries the inflammation referred to is commonly limited to the adhesive process, —to the closure of the diseased portions,—occasioning coagulation of the affluent blood up to the nearest free branch. In the veins, on the contrary, the inflammation advances in the direction of the heart, as far as the irritative fluid pursues its course, and, unless speedily and completely confined by *sequestration,* inevitably leads to the well known fatal consequences.

Phlebitis may originate not only from traumatic and from purely septic causes, but likewise from *rheumatic* affections. It is then less prone to purulent formation than to plastic exudation, remaining stationary at the adhesive stage, and restricting itself in many cases to coagulation of the fibrin within the vessels.[1]

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usually begins above and then descends, the reverse happening in phlebitis. When, however, I was myself afflicted with phlebitis, the swelling originated at the upper part of the saphena, and gradually proceeded downwards.

[1] This view is not yet fully corroborated by facts. However, besides the first and third cases in an essay by Baron (sur la Coagulation du Sang dans l'artère pulmonaire, Arch. gén., Mai 1838), I may instance that of a robust youth at the Leipsic hospital, who, after taking severe cold, became seized with wandering pains and with a fixed and deep-seated pain in the sacral region. Violent dyspnœa ensued, and he died very shortly afterwards. On dissection, nothing was discovered except inflammation of both hypogastric veins, which were blocked up with solid, adherent, and stratiform fibrinous plugs; similar formations were found within the branches of the pulmonary artery of the right side.

Finally, it should not be overlooked that phlebitis is very often developed under *epidemic* influence, in which respect it appears nearly allied to erysipelas. This accounts for the frequent mortality which manifests itself at certain periods among hospital patients who have undergone operations or sustained injuries,—perhaps, likewise, for many epidemics of puerperal fever.

Subjoined is a brief notice of the inflammation of certain individual veins, which, owing to the peculiarity of its occurrence and the concomitant symptoms, appears to merit special consideration.

Inflammation of the *sinuses of the dura mater*, anatomically considered, closely resembles ordinary phlebitis. The same layers of fibrin fill up the sinuses and adhere to their parietes: the same soft masses of coagula speckled with gray, and of true pus are discovered. The coagulation of the blood extends in the same manner to the branches of the veins, and to the vessels of the brain and of its membranes. The inflammation and its products proceed towards the trunks, and finally, through the jugular vein, to the heart. Commonly the sinuses of one side only are affected, even though a longitudinal sinus may be implicated. Death ensues very speedily, amid the phenomena of functional disturbance of the brain; considerable quantities of serous fluid are found accumulated beneath the arachnoid, and in the ventricles; the adjunct cerebral substance is softened; the whole encephalic mass gorged with blood. Plastic exudation is frequently found to have taken place between the membranes. Inflammation of the sinuses is mostly a secondary affection, depending upon jugular phlebitis; upon purulent exudation of the arachnoid;[1] upon caries of the cranial bones;[2] upon suppressed porrigo; upon scrofulous ulcerations at the occiput; or upon cerebral softening,[3] from the irritation caused by splinters of fractured bones of the skull.[4] It may, however, occur as a primary disease.[5]

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[1] Two cases observed by myself, one by Gendrin (loc. cit.), and another by Ribes (Rev. Méd., vol. iii.)

[2] Arnott, Abercrombie, and many others.

[3] Three cases by Tonnelé (Journ. Hebdomad., vol. v, Févr. 1829.)

[4] Schmucker (Chir. Wahrnehmungen, vol. i, p. 160.)

[5] Cruveilhier, livr. viii, pl. iv.

Inflammation of the *portal vein* has been observed twice by Bouillaud,[1] twice by Renaud,[2] and once by Dance,[3] once by Balling,[4] twice by Schönlein,[5] once by Mohr,[6] and once by myself, as a sequel of typhus. It is attended by icteric phenomena, by diarrhœa, and vomiting, often of blood. If the disease be chronic from the commencement, or become so in its course, there is considerable emaciation, together with ascites and general dropsy, especially if the inflammation reach the inferior vena cava. The obstructed circulation seeks to re-establish itself by preternatural distension of every available anastomosis. The veins of the portal system are found generally gorged and dilated; the spleen enlarged. Organic change of the liver is, however, not observed in every instance, even when the disease has been acute.[7]

We have still to advert to the recorded cases of inflammation of the *umbilical vein* in new-born infants.[8] The symptoms during life were jaundice, vomiting, diarrhœa, and erysipelatous inflammation surrounding the umbilicus. After death, all the signs of inflammation were discoverable in the umbilical vein, occasionally extending to the vena portæ and to the hepatic veins, with more or less diffuse peritonitis, and in some instances with the usual secondary changes, in other organs, that result from the commingling of pus with the blood.—It is remarkable that the liver in no instance appeared to be the seat of any such changes.

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[1] Arch. gén., vol. ii, Juin 1823.

[2] Journ. Hebd., vol. ii, No. 24, and Rev. Méd. 1839.

[3] Twentieth case.

[4] Loc, cit. p. 310.

[5] Baczynski Diss., Turici, 1838) and Güterbock (Klin. Vorträge v. Schönlein, 1842.)

[6] Centralzeitung, 1840, No. 29.

[7] Compare a case cited by Balling (from Fizeau) of inflammation of the hepatic veins.

[8] One by Osiander (Neue Denkwürdigkeiten, vol. i, p. 57), two by the elder Meckel (given by Sasse de Vas. Sanguif. Infl., Hal. 1797), five by Duplay (l'Expérience, Dec. 1838), and two by Schöller (Neue Zeitschr. für Geburtskunde, vol. viii, fasc. ii.)

### SECT. II.—OBSTRUCTION AND OBLITERATION OF VEINS, INFLAMMATORY—MECHANICAL—CACHECTIC.

The plugs developed by inflammation within veins are either removed within a short time by resolution, or become to a certain extent organized, and operate as permanent impediments to the circulation. Sometimes, however, they are not the immediate products of disease of the venous membranes, but result from pressure exerted upon the vessels by tumours of various kinds, to the extent of retarding at first, and in the end completely interrupting the sanguineous current. The blood then coagulates, irritates after the manner of a foreign substance, and induces a slow phlebitis, which, in time, leads to a thorough obliteration. Numerous examples of this kind have been recorded, and must necessarily have come under the personal observation of all who have been much engaged with morbid anatomy. To effect this coagulation, however, particular circumstances must co-operate with the pressure above alluded to. For we may observe, for example, the *gravid* uterus so to compress the one or both of the iliac veins as to cause œdematous swelling, varices, and stagnation of the blood, without coagulation taking place; whilst it is readily occasioned by the pressure of a uterus enlarged by cancerous degeneration. In a word, the plug-formation from mechanical causes is only to be met with in weakly and cachectic individuals, exhausted or rendered paralytic by the effects of other diseases. Thus, aneurism very frequently gives rise to the obliteration of contiguous veins.

Instances occur of the complete obstruction of veins by medullary fungoid excrescences, which either form between the membranes, or perforate the latter and vegetate within the canals. This is very frequently observed in the branches of the vena portæ, in cancer of the liver, whence closure not only of the trunk of that vessel, but likewise (from within the hepatic veins) of the vena cava inferior may ensue. In the case of a considerable medullary-fungoid tumour of the lymphatic glands of the neck, I found the right jugular vein obliterated (probably from compression) so that no trace of that blood-vessel was discoverable.

On the other hand, the fungus had penetrated into the subclavian vein, and from thence into the vena cava superior, completely filling them up, and projecting in a jagged manner into the right auricle of the heart. Unfortunately I was compelled to make the examination under circumstances which prevented the collateral circulation being closely examined.

H. Weissbrod[1] has minutely described a similar case, equally remarkable for the symptoms during life, and for the appearances on dissection. The same writer has moreover adduced the rare example of an adhesion of the pulmonary veins of the right side, brought on by pressure, from the descending cava having been choked up with a cancerous growth. The closure was here so complete, that the above-mentioned vessels could not be traced into the collapsed, dense, and airless lung;—for the right bronchus was also found closed. In a case of cancerous affection of the lungs I found one of the pulmonary veins thoroughly obstructed with the cancerous substance, so that the morbid excrescence protruded into the left auricle of the heart. A considerable contraction of the left pulmonary veins was observed by Townsend.[2] The most violent dyspnœa, to be mitigated for a short space of time only by repeated blood-lettings, was followed by death through asphyxia. The left lung being cut into, blood gushed out; all the branches of the pulmonary veins were distended to at least four times their natural size; their trunks had become transformed into dilated sacs, and, immediately before their entrance into the auricle, a layer of *tubercle*, an inch thick, was found imbedded between their membranes, so as scarcely to admit of a probe being passed into the auricle.

Ossification,[3] though of exceedingly rare occurrence in the membranes of veins, may now and then render these vessels impervious. The same effect may be produced by calcareous concretions, either those of a cylindrical form sometimes seen obstructing the uterine veins, or the common round phlebolithes or veinstones.[4] Closure of this description is, however, of no moment, since it hardly ever affects any but the smaller venous branches.

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[1] Obs. duæ Venam cavam desc. tang., Monach, 1831.

[2] Dublin Journ., Jan. 1833.

[3] Examples in Puchelt (Das Venensystem, &c., p. 205), and in the elementary works of Voigtel and Otto.

[4] Vide Phlebectasis.

So many channels are generally open to the venous blood, that a permanent interruption to the circulation of a part can only be accomplished by the obliteration either of most of the venous branches of a member of the body, or else of one of the larger venous trunks. Such interruption manifests itself principally in the occurrence of dropsical swellings. Thus, œdema of the upper and lower extremities is observed as a consequence of the stoppage or compression of their principal veins; ascites from an impervious condition of the vena portæ, a frequent result of cancer of the liver. Bouillaud [1] has shown that the greater number of partial dropsies are referrible to an impediment more or less complete in the veins,—Zacharias Platner,[2] having *previously* ascribed œdematous swellings to the impeded reflux of venous blood. Numerous observations, ancient as well as modern,[3] might be cited in corroboration of this view, but direct proof was furnished a hundred years ago, by Lower's experiments upon the tying of veins.

Even this dropsy, consequent upon the obliteration of veins, is susceptible of natural cure, a dilatation of the anastomoses serving to establish a *collateral circulation,* which nullifies the impediment created within the normal channel. In this respect, the closure of the vena portæ is confessedly the least easily compensated for, and incurable dropsy appears to be its necessary consequence, although the roots of the portal vein are in several places connected with the veins of the general system, so that, through dilatation of these branches, a total stagnation is prevented. Besides this, the blood makes way for itself, through channels traversing the mass by which the vein is clogged;[4] as Reynaud noticed even in the portal vein.—If, on the other hand, one of the venæ cavæ has become impervious, the embarrassment thence arising is more easily counterbalanced. The ascending cava, in particular, is replaced by the dilatation of the azygos and hemi-azygos, by the anastomoses of the epigastrica with the mammaria interna, and of the subcutaneous abdominal with the axillary veins.[1] In the case of obliteration of the descending vena cava,[2] especially where the mouth of the azygos is at the same time closed, the auxiliary circulation is more complicated. It is established, partly through the plexuses of veins of the spinal canal, which are connected both with the subclavian and with the hypogastric veins; partly through the anastomoses of the phrenic veins, which inosculate with the vena cava inferior, and probably with the great coronary vein of the heart, as shown in Reynaud's case,[3] partly through the axillary veins and the mammaria interna, which, through the medium of the epigastrica and of the circumflexa ilei, are enabled to transmit their blood to the ascending cava. Instances of dropsy have been recorded, in which the enormous distension of these last mentioned subcutaneous vessels has led to a recognition of the closure of veins, even during life.[4]

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[1] Arch. gén. vol. ii, Juin 1823.

[2] Institut. chirurg. ed. nov. Lips. 1783, par. 729, 730.

[3] Vide, amongst others, Corbin (Arch. gén. 1831.)

[4] Vide pages 25 and 26.

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[1] A very good description of this collateral circulation is furnished by Gély (Gaz. Méd. de Paris, 1840, No. 45) in an instance of inflammatory obliteration of the femoral vein and ascending vena cava.

[2] Hodgson knew of no example of complete closure of the descending vena cava; Puchelt and Otto, however, cite instances from older writers. Moreover, Otto (Seltene Beobacht ii, p. 64), Deckart (Diss., Berol. 1823), and Martin Solon (Arch. gén. 1836, vol. x, p. 296), found this vein closed by the pressure of aneurisms. J. Reid and W. Thompson (Edinb. Med. and Surg. Journ., Apr. 1835) met with its spontaneous obliteration, and I have already mentioned two cases of obstruction of the descending vena cava by carcinomatous growths. Bouillaud (Arch. gén., Juin 1833.) Stannius, in his excellent monograph (Ueber die krankhafte Verschliessung grösserer Venenstämme—on morbid closure of the large venous trunks—1839) has recorded a very remarkable case in which not only the descending vena cava, with its principal branches, but likewise the greater portion of the ascending vena cava had become closed by the gradual deposition, in layers, of a yellowish-brown mass.

In the above work sixty-eight cases, since observed by himself, are referred to, and inflammation shown to be the most frequent source of obliteration of the veins. He remarks very justly that the collateral venous circulation, though facilitated by the numerous anastomoses, finds some impediment in the valvular apparatus of the veins, which, though apparently sometimes overcome, in general probably subjects the venous collateral circulation to the laws governing that of the arteries; that is, the circulation being so obstructed, the blood would at first make its way through the smallest anastomoses, unprovided with valves, and by and by cause some of them to dilate.

[3] Journ. Hebd. 1829

[4] Thus (before Bouillaud) by Kreysig.

## SECT. III.—PHLEBECTASIS.—DILATATION OF VEINS.

The dilatation of veins is one of the most frequent morbid changes of structure occurring within the human body. It manifests itself in innumerable cases as a morbid predisposition, yet not unfrequently as an essential disease, forming most commonly a source of abiding annoyance and suffering; and if not fraught, strictly speaking, with imminent danger to life, still capable, under particular circumstances, of operating as the immediate cause of death. Three varieties of disease equally frequent in occurrence, are referrible, anatomically speaking, to the dilatation of veins: the properly so called *varicose veins,—varicocele,*—and *hemorrhoids.* It would be discordant with sound pathological views to regard the term phlebectasis as fully expressive of the characteristics of these three forms of disease. Yet it cannot be denied that they possess no other anatomical peculiarity in common, which distinctly indicates the morbid process in which they originate. At all events, the sequel will show that there is no inconsistency whatever in classing these three forms of disease under one head.

All three have their chief and common source in a peculiar habit of body, a morbid predominance of the venous system, which manifests itself through the intervention of influences at once mechanical and dynamical. This uniformity of cause in constitutional disposition cannot certainly be substantiated by the appearance of the three affections conjointly in one and the same individual; because it often happens that the full development of this disposition in one particular locality, prevents its outbreak in another. Thus, Landouzy[1] rarely witnessed hemorrhoids or varices in persons afflicted with varicocele. Brodie, again, observed[2] that while hemorrhoids affect by preference persons belonging to the affluent classes, varices are comparatively more frequent amongst the lower orders.

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[1] Landouzy, du Varicocele et de la cure radicale de cette affection. Paris, 1838.

[2] Lectures on varicose veins and ulcers (Med. Gaz. 1837, Oct. p. 184, Nov. p. 264; 1835, Feb. p. 742.)

Far from calling in question, as Landouzy does, the pathogenetic connexion between the two diseases, we may rather affirm that they are known from experience not entirely to *exclude* each other. I am, indeed, disposed to conclude, from my own observations on dead bodies, that hemorrhoids more frequently coexist with varicose veins, than experience among the living has hitherto justified us in supposing. In post mortem investigations, examination of the rectum and anus (too oft neglected) cannot, for this reason, be too strongly inculcated. My own experience, however, tends only to confirm what has been already stated, namely, that in cases of well marked varicose veins of the leg, or of extensive varicocele, hemorrhoids are only in a very slight degree developed, and inversely.[1] The same reciprocal relations exist between hemorrhoids of the veins of the rectum, of the bladder, and of the vagina,—the one always occurring in a limited manner, whenever the other is fully developed. And yet they are evidently all manifestations of one common habit or predisposition; and each, both in its totality and in its often regular periods of exacerbation, a critical effort of nature. Further evidence is afforded by the hereditary character of the several forms of phlebectasis, and especially by the fact that the offspring of a parent, subject to one form of the disease, is liable under propitious circumstances, to become affected with either of the other forms.

It is by no means unimportant to consider the respective periods of life at which the several varieties are wont to appear. Varicocele most frequently commences between the age of puberty and the thirtieth year; for, although often met with at a later period, it is then commonly a residuary affection; not one of recent origin. Varicose veins of the leg are for the most part developed from the twenty-fifth year upwards, persist during manhood, and decline in old age.[2] Hemorrhoids of the rectum usually set in from the twenty-fifth year upwards, outlast the prime of life, and are often replaced, after the period of the grand climacteric, by vesical and vaginal hemorrhoids,—occasionally disappearing altogether, or nearly so, in very advanced age. Exceptions (particularly instances of hemorrhoids in children) are adduced by various authors, but cannot of course invalidate the general rule.

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[1] It is, however, to be observed that the separate occurrence of dilatation of the veins of the spermatic cord is far more frequent than that of hemorrhoids. This is perhaps to be explained in the independence of all parts of the sexual apparatus in their physiological and pathological relations.

[2] Briquet, Mémoire sur la Phlebectasie (Arch. gén. de Méd. vol. vii, p. 200.)

In studying the subject anatomically, we find phlebectasis passing through various stages in the course of its development, and, according as the predisposition is less or more intense, either remaining stationary, or advancing to its highest point of development, and spreading to the utmost extent of its limits.

The individual stages or degrees in question have been minutely described by Briquet,[1] and the description confirmed by Andral.[2] The latter, indeed, presents us with several additional varieties,[3] the fifth of which might with more propriety be classed as a subdivision of the rest, whilst the sixth, strictly speaking, does not belong to phlebectasis at all.

In persons affected with a morbid preponderance of the venous system, we first of all observe an undue prominence of the veins of the skin. These appear in dense nets of branches, remarkable for their diffuse distribution, and are generally turgid with blood, or liable to become so from the slightest mechanical or dynamical causes,—like what, under ordinary circumstances, would be the effect of violent and prolonged muscular exertion. In this condition of the veins, their coats have not undergone any absolute change, being every where proportionate to the width of the caliber. The vessels are not more than usually tortuous, and cannot as yet be called morbidly altered.

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[1] Loc. cit.

[2] Puchelt (Vide Das Venensystem in seinen krankhaften Verhaltnissen—the venous system in its morbid relations—Leipzig, 1818) was the first to point out the necessity of distinguishing certain varieties of venous dilatation, and assumed four species; but his classification has too little foundation in anatomy to make it available here.

[3] 1. Simple dilatation, without concomitant change of any other kind. 2. Equable general or partial dilatation, with attenuation of the membranes at the dilatated part. 3. General dilatation, with thickening of the membranes. 4. Partial dilatation, with thickening of the venous membranes. 5. Dilatation of the veins, with development of imperfect septa within their caliber. (I believe I shall be able to prove in the sequel that these so-called septa are not of new formation.) 6. The same condition, with cribiform perforation of the venous coats, and communication with the diseased cellular tissue,—erectile tumours. (See the Appendix to Artericctasis. Andral, Précis d'Anat. Pathol.)

After a while, however, the veins become permanently dilated, and more distinctly prominent, an occurrence more frequent in elderly than in young persons. This is brought about by a reinforcement of the fibrous texture of their external coat, in the shape of an accession of conspicuous transverse fibres. Meanwhile the internal membrane remains unchanged in structure, merely displaying numerous lines or superficial furrows running lengthwise; and the vessel still maintains its natural course, not assuming a more sinuous, but rather, if anything, a straighter direction than before. It does not collapse, when cut through, but remains patent, and is distinguishable from the arteries by its colour, which is of the same pale red as the fibro felt-like texture constituting the normal external membrane of a vein. The valves remain unaltered. In this condition the saphena vein is frequently found in old persons: so likewise are certain branches of the vesical plexus, whilst other branches manifest still further changes.

In the greater number of instances, however, the external membrane of the vein is not thickened, but, along with the other membrane, undergoes considerable attenuation, in proportion as the vein becomes more and more dilated. Conformably with their irregular disposition, the intermediate fibres give way unequally, allowing the internal membrane to jut out in sac-like protrusions, and to establish so many irregular, constricted, pear-shaped, and often in appearance pediculated, tumours. At the commencement of some of the smaller branches, the membrane thus forms pouch-like dilatations, or forces itself betwixt the longitudinal fibres of the external membrane in lengthy protuberances, which exceed in circumference that of the vein in its natural state; or it may perhaps distend cylindrically and pretty equably for a considerable length the intermediate fibres before alluded to.

Meanwhile the valves become attenuated and pulled asunder transversely, so as to be rendered useless. In many instances, they become partially or wholly obliterated, or are torn into shreds, or destroyed as far as their free border, which then runs across the diameter of the vessel, like a filament or band attached by the two extremities to the internal membrane.[1] The veins now appear elongated, and their course very tortuous. Their canal is wide and narrow by turns, now simply deviating, now throwing out pouch-like appendices, then again dilating into irregular shaped cavities, until the whole vessel has assumed an unwonted aspect, reminding us at one moment of the seminal vesicles, at another, of the convolutions of the intestines.

This general sketch is applicable to almost every variety of phlebectasis, subject, however, in the special description to certain modifications, necessarily resulting from the structure, function, and connexion of individual organs.

We shall begin the special inquiry with the consideration of *varices of the leg.* Where the predisposition exists, they make their appearance after the twenty-fifth year—sometimes earlier; more frequently in men than in women;[2] and mostly as a consequence of habitual toil, pursued in the erect posture and coupled with continued muscular exertion of the lower extremities.[3]

It is remarkable that *in men,* the dilatation usually arises from the trunk, or the principal branches of the saphena; *in women,* from the minutest twigs of the cutaneous veins. The latter then become conspicuous as extremely minute but dense, dark violet-coloured ramifications, sometimes unequally dilated; commonly occupying the inner side only, but occasionally overspreading the whole of the leg, and gradually involving the deeper seated branches. It is, however, in rare instances only that we observe varices affecting the whole leg: it is more usual to find only a few branches diseased, commonly those of the internal saphena; and again of these, most frequently,—perhaps solely—one or two branches which pass obliquely downward from the internal condyle of the tibia towards the lower portion of that bone. The affection is not always symmetrically developed in both legs, but often confined to one, perhaps, in the majority of cases, the *right* leg.[1]

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[1] This condition of the valves is the source of dangerous hemorrhage from the upper portion of a divided varicose vein. (Vide Velpeau, Traité d'Anat. Chir. 3e. éd. p. 104.

[2] Briquet found, amongst 258 males, 71; amongst 483 females, 42 affected with varix.

[3] In general, they are slowly developed; but now and then rapidly, as shown by Briquet and by Brodie.

In rarer instances there exist considerable varices of the trunk and branches of the femoral portion of the saphena, while the veins of the leg remain sound. Through the medium of numerous and not inconsiderable anastomoses, these dilated portions of the saphena, and of its tributaries, are connected with the deeper seated veins accompanying the arteries; whilst amongst each other, they are woven into a net-work of vessels, which ramify and inosculate with each other in the most complex manner. The cellular texture surrounding the varices is often thickened, the adipose tissue displaced, and their cells infiltrated with a dull white, pellucid, lardaceous serum. Within this substance the veins lie imbedded, either in furrows and loosely attached, or else firmly coalescing with it.[2] Under these circumstances the section of the vein, when divided, remains gaping. Amussat found the lymphatic vessels within the thickened cellular texture, adjacent to varices, considerably dilated. Where the mischief is at all extensive, the circulation in the leg is so much embarrassed that a very distressing, painful œdema is the result. The occurrence of this is at first limited to the evening; by-and-by, however, it becomes permanent, and is, under particular circumstances, accompanied by erysipelatous inflammation of the skin. The varicose knots sometimes project so far as to displace the superficial cellular texture. They then coalesce with the external skin, and attenuate it in proportion as the dilatation advances, until at last the varix bursts, giving rise to violent and often fatal hemorrhage. Perforation is occasionally preceded by the formation of ecchymosis, and by a circumscribed sub-inflammatory process.[3] Varicose veins frequently inflame, but the inflammation is generally partial, and rarely exceeds the adhesive grade. In such instances the blood coagulates within the dilated portions; the plug either consolidates and effects a complete or partial obliteration of the varices,[1] or else gradually liquefies, and that often in a very short space of time. Occasionally, however, the inflammation goes on to suppuration, and proves fatal by a general infection of the blood. An attempt is made to cure the disease by calling forth, by artificial means, an adhesive phlebitis. This, however, seldom succeeds; for, in most cases, either the inflammation advances to the highest destructive grade, or else obliteration is imperfectly, or not at all achieved. Simple division or tying of the vein is insufficient; the latter, especially, because the thread effects no division of the internal membrane, and consequently no adequate adhesive inflammation.

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[1] This is Briquet's opinion.

[2] It is for this reason often very difficult to disengage varicose veins with the scalpel for the purposes of examination; yet, with care, it may be accomplished, especially in dropsical subjects.

[3] In rare instances there occurs a periodical rupture of varicose tumours, supplementary of some other suppressed hemorrhage. (Franck von Frankenstein, Bordeu, Briquet.

The alterations which the blood undergoes in varicose veins have not yet been fully ascertained. The circumstance of its having been usually found of a florid red, resembling arterial blood, and occasionally seen to issue in jets from the incised vein, has led to the inference that a free communication must exist between these veins and the arteries; but neither observation nor experiment warrants the conclusion. It is, however, probable that the blood within the varices suffers, from long stagnation, certain changes which may not be without influence upon the remainder of the sanguineous mass. Coagula of various shape and extent frequently occur, and may, for the most part, be reckoned products of a sub-inflammatory condition. Phlebolithes, or veinstones within the varicose dilatations of the leg, are amongst the rarer phenomena; I have always found them compressed and flattened. Their formation will be subsequently explained.

*Varicose ulcers* are amongst the most frequent consequences of phlebectasis. They are the result either of circumscribed phlebitis, attended with suppuration of the neighbouring parts, or are developed through some external and accidental source of irritation, and eventually kept up by a morbid condition of the cutaneous texture. Ulcers of this nature often spread very diffusely; several are met with at the same time; they are surrounded by dilated veins, which sometimes participate in the progressive ulceration, and cause violent hemorrhage.

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[1] Hodgson and others.

Owing to the constant irritation, single branches in their vicinity become attacked with phlebitis. In short, just as these ulcers are themselves a result of the phlebectasis, so do they, in their turn, tend unceasingly to augment the original evil.

Varices occur not only in the lower but also in the upper extremities, and in various other parts of the body besides. The case of J. L. Petit, in which a large varix near the bend of the arm was opened for blood-letting, is well known. Two instructive cases are related by Warren,[1] the one of a boy eight years of age, who, through violent muscular exertion, contracted a varix between the shoulder and the elbow; the other that of a West Indian gentleman, upon both of whose arms varices of an inch long, were developed in quick succession, as the sequel of a tropical fever.[2] Brodie has seen varices along the arm and the right side of the thorax, induced by the pressure of a glandular tumour upon the right subclavian vein. Thus we find that here the same causes determine the same disease as in the leg, but in an inverse proportion as regards frequency. Is the slower ascension of the blood, and consequently augmented pressure of that fluid upon the veins of the lower extremities alone in fault; or is the quality of the blood in these parts, further removed from the central organ of circulation, to be taken into account as a co-operating cause? A decisive answer cannot at present be given to this question.

Phlebectasis of the veins of the spermatic cord is commonly termed *varicocele* or *cirsocele.* Its frequency is probably exaggerated, when it is asserted that out of every hundred military recruits, sixty are afflicted with this evil.

The affection is most frequently developed during puberty. According to Landouzy, out of forty-five cases, thirteen set in between the 9th and 15th; twenty between the 15th and 25th; and three between the 25th and 35th years of age. Dynamico-mechanical irritation of the organs of generation, during these periods, is generally the exciting cause. Onanism, immoderate sexual intercourse, riding, dancing or walking in excess, are to be considered as principally instrumental in originating the disease. Its more frequent occurrence on the left side is a remarkable fact; this has been attributed partly to the more constrained progress of the blood (the left spermatic vein entering the renal at *a right angle*, the right passing *obliquely* into the vena cava), partly to the pressure exercised by the sigmoid flexure of the colon with its solid contents, upon the left spermatic vein. To these should perhaps be adjoined the very general practice of carrying the scrotum upon the left side, whereby the left testicle and spermatic cord are obviously more compressed than the right, and at the same time exposed to a degree of warmth favorable to the production of the evil. At all events it is certain that the more numerous and more marked instances of varicocele affect the left side; for although those of the right side are by no means rare, they are mostly stationary, never attaining a great bulk, nor requiring surgical interference. Out of 120 cases operated upon by Breschet, one alone was on the right side. Varicocele, when forming independently of development of the sexual function, namely in old age, is generally referrible to the pressure exercised by tumours within the abdomen, to hernia, to the use of inappropriate trusses, tight breeches, &c. Under all circumstances, varicocele seems to occur more frequently in warm than in the colder climates.

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[1] Surgical Observations on Tumours, p. 432. Boston, 1837.

[2] Compare Cruveilhier, livr. xxiii, pls. iii, iv, livr. xxx, pl. v, cases in which numerous phlebolithes had formed simultaneously in the dilated veins.

The disease commences with dilatation of the numerous minute venous twigs of the spermatic cord, situate betwixt the external abdominal ring and the testicle. This dilatation is at first general and cylindrical; the external tunic of the vein becomes simultaneously hypertrophied, as above described; the vessels implicated often dilate to the width of a goose quill, and are constantly gorged with blood. Presently their course becomes exceedingly tortuous, and pouch-like enlargements of irregular shape form at intervals, where the external tunic happens to be unequally thickened. In proportion as the venous web increases in bulk, it sinks along the vas deferens to beneath the testicle, so as to incase it almost entirely. The testicle now begins to waste away, its veins proceeding to dilate at the expense of its parenchyma. It is doubtful whether the same morbid condition spreads upwards to the vessels within and beyond the inguinal canal: my own observations are against such an assumption. Circumscribed phlebitis, of a very painful character, now and then supervenes, but seldom spreads widely, and probably never oversteps the adhesive grade. The original malady is frequently complicated with hydrocele. Rupture of varicose veins of the spermatic cord determines the majority of instances of hæmatocele. In many cases the scrotal veins dilate simultaneously, but never to a great extent. Phlebolithes have occasionally been found within the vessels of varicocele.[1] I have once met with them in dilated veins of the scrotum. In its simple relation to the corporeal organism, varicocele is, for the most part, to be looked upon as an isolated disease, almost exclusively local in its tendencies, and very rarely productive of organic mischief in other parts. It, however, assumes a serious character, and in fact derives its true import, from the ascendancy which it is known to acquire over the moral faculties of the patient. For it is a truth well attested by experience, that varicocele when fully developed (at the cost of the parenchyma of the testicle) gives rise to a melancholy which not unfrequently ends in suicide. We may, however, observe that this aberration of mind has its source less perhaps in the disease itself, than in the previous excessive abuse of the sexual function, for which it is the penalty.

We come, in the last place, to the most important of all the varieties of phlebectasis—*hemorrhoids.* Cullen, Chaussier, and Recamier have considered *hemorrhoids* or *piles* to be nothing more than extravasations of blood within the cellular texture; and more recently Gendrin[2] and other pathologists have sedulously endeavoured to prove that veins have no part in their formation. Other inquirers, struck with something incongruous in the assumption, and yet unwilling to revert to the ancient, simple explanation, have adopted the opinion of Delpech and Cruveilhier, namely, that they are tumours of erectile tissue. Numerous researches, however, instituted according to various methods, have induced me to take up the older view, and, along with Puchelt, Hodgson, Andral, Lobstein, R. Froriep, and many others, to regard all hemorrhoidal tumours as dilatations of views. That so many eminent pathologists should entertain a different opinion is due, partly to the inherent difficulty of the subject, partly to the circumstance that the rectum is seldom examined after death. In looking back historically to the origin of this unwillingness to refer the real seat of hemorrhoids to the veins of the rectum, we are not a little astonished to find it in Harvey's discovery of the circulation. The fact is, however, clearly attested in certain Essays published by Langguth, at Wittemberg, and in Santorini's 'Opusc. Med.' Rotterd. 1719; (Opusc. tert. page 150.) Medical men would not believe that hemorrhage, yielding often very florid blood, could have its source in veins, whose function of centripetal conveyance had been clearly recognised. Phlebectasis, at the extremity of the *rectum*, assumes a peculiar character. It is principally very small venous twigs that become varicose. These little veins crowd together at the margin of the anus, and are in such intimate communication with each other as to constitute a web that might pass for a very close approach to erectile tissue. Several contiguous branches dilating simultaneously constitute what are called hemorrhoidal knots (or piles) which, sometimes of unequal size, surround, like a string of beads, the margin of the anus,—sometimes externally, sometimes internally, and only in rare instances above the external sphincter. These dilated veins generally belong rather to the submucous coat than to the subjacent cellular texture; at any rate their dissection from the latter is a work of no great difficulty, whilst I have never been able to detach them in a connected state from the mucous membrane.

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[1] The term cirsocele has been by some writers restricted to phlebectasis of these veins, and varicocele to that of the veins of the spermatic cord. Velpeau, in his Traité d'Anat. Chir., makes this distinction.

[2] Gendrin, Traité Philos. de Méd. Prat. Paris, 1838. Vol. i, p. 332.

On minutely examining such an hemorrhoidal tumour, we find it to consist of an indefinite number of separate cells, which are generally formed by the dilatation of several branches of veins, and are connected together by layers, more or less thick, of a reddish, and, in some instances, indurated cellular texture. These cells have commonly an irregular spherical shape, and are lined with the internal venous membrane sufficiently attenuated, but, in tumours not inflamed, perfectly smooth. Into each individual compartment or cell, several, usually very minute, twigs enter,—forming media of communication with the larger branches, and also with the neighbouring cells. R. Froriep[1] is satisfied that this delicate framework of the cells is an immediate continuation of the internal membrane of the veins. Provided the hemorrhoidal tumours be neither inflamed nor clogged with coagula, injected fluids will promptly penetrate from one cell into another, and into the larger venous branches. In a large number of hemorrhoidal tumours I have been able to trace this direct communication very distinctly, by the aid of inflation. The reason why Chaussier and Recamier failed to inject the tumours from the internal hemorrhoidal vein, and yet easily filled the connecting cellular texture, by injecting the arteries, was, that the tumours were in a state of erethism, and replete with coagula, at the time of experiment. Brodie expressly states that piles, *not* in a turgescent state, fill amply, on injecting the inferior mesenteric artery.

Most piles, and indeed all at the first, are sessile upon a broad base. When, however, they are of long standing, and have suffered many attacks of periodical turgescence, they frequently protrude during the efforts to expel the contents of the rectum, and, becoming constricted at the margin of the anus, assume a pedunculated aspect.[2] This is owing to the following circumstances: every anatomist is familiar with the very regular, natural folds and sinuses (lacunæ) situate at the extremity of the rectum, and within the circuit of the external sphincter. The piles almost always form upon these prominent folds, or beneath the sinuses, and by projecting in a circle around the latter, enlarge them into deep pouches. In the act of defecation, the excrements are forcibly driven into these pouches, and the piles themselves thereby thrust forward beyond the margin of the anus; nor is it at all improbable that the prolapsus of the intestine, so common in persons subject to hemorrhoids, originates from the came cause. As the disease makes further progress, however, not only do the smallest veins dilate more and more, but varices of the larger veins form in like manner, constituting those piles which are noticed by several authors as occurring high up within the rectum.

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[1] Chir. Kupfertaf. pl. 113, 114.

[2] The whole margin of the anus is found occasionally protruded and constricted in this manner. (See an extraordinary example in Cruveilhier, livr. xxv, pl. iii, fig. 1.)

All the anal hemorrhoids which I have examined bore essentially the character above given, and differed from each other only in respect to the size and number of venous branches engaged in the dilatation.[1]

At certain periods, often tolerably regular in their recurrence, the peculiar phenomena of *turgescence* set in, and lead either to an alleviating hemorrhage or to a painful and obstinate swelling of the piles. Some patients continue, during a long series of years, to experience such periodical attacks, little varying in intensity; while to others, each paroxysm is fraught with an augmentation of suffering; inflammation of the tumours being followed, step by step, by the development of abscesses within and round about; by the formation of scars and of stricture at the margin of the anus; by fæcal fistulæ and by prolapsus ani. The anatomical peculiarities of the tissues surrounding and forming the anus, together with the almost uninterrupted sources of violent irritation arising from the function of those parts, readily account for a limited hemorrhoidal phlebitis leading to formidable local mischief. At the period of turgescence the vessels of the hemorrhoidal tumours attain their greatest possible distension. The venous cells fill to repletion, the minutest extremities of the arteries appear in a very close but delicate network upon the intervening laminæ of cellular tissue, which become infiltrated with plastic matter and straightway harden. Under such circumstances a copious gush of florid red blood sometimes issues from the entire dark red and shining surface of the mucous membrane. A coagulation of the blood takes place simultaneously within the venous cells, extending from thence to the veins themselves. The clots thus formed again liquefy, one or two alone remaining in one or another of the numerous cells of a hemorrhoidal tumour, perhaps to undergo in the sequel a further change; namely, conversion by calcareous deposition into a phlebolithe or vein-stone. Or an adhesive inflammation is established throughout the entire hemorrhoidal tumour, ending in its gradual but definitive reduction, so that, at the next paroxysm, its place becomes occupied by neighbouring piles. In other cases the cellular tissue of the pile, together with its mucous integument suppurates, and a smaller or greater number of circumscribed little apertures form, which penetrate to within the venous cells. A little purulent matter will then escape by the anus, and inconsiderable hemorrhage ensue,—recurring at intervals until the irritative process has subsided. I am not acquainted with any instance of a circumscribed phlebitis, like the above, spreading to the larger veins, and thus leading to a fatal issue: examples are, however, not wanting of such being the result of operative interference, more especially of tying piles.

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[1] The writers alluded to explain the origin of hemorrhoidal tumours thus: "Blood effused from the extreme ends of the arteries becomes encysted by and incorporated with the cellular texture; this, with the cooperation of the mucous crypts, gives rise to saccular hemorrhoids: if, on the other hand, the cellular texture becomes engaged in a process of active hypertrophy, and its cells enter into intimate communication with the extremities of the blood-vessels, the fungoid form becomes developed." Here all analogy as to extravasation of blood in other parts is set aside, and imaginary results are substituted for facts. Recent investigation (see Henle) has, moreover, shown the minute structure of cellular tissue to be very different from what must have been presupposed by the framers of the above theory.

The occurrence of hemorrhoids *in the bladder* and its appurtenances has been denied by many, especially by those who consider the term hemorrhoidal tumours to signify extravasations or erectile swellings, either of which will indeed be sought for in vain in connexion with that viscus. The affection consists in a dilatation of the veins of the prostatic and vesical plexuses;—as far as the latter is concerned, for the most part of the branches about the neck of the bladder and on either side of the seminal vesicles, external to the muscular coat. The branches running beneath the mucous membrane very rarely dilate, and if any do, it is those about the neck of the bladder and the commencement of the urethra. This happens principally with men, and, as it would seem, at an advanced period of life, after the 45th year. I have not yet met with any recorded instance of hemorrhoids forming at once within the region of the bladder, without previous disease of the rectum. In females this variety seldom occurs; the disease, in their case, being seated, if not in the rectum, almost invariably in the veins of the upper portion of the vaginal walls. Still I have examined several aged females in whom there certainly were piles, though small ones, at the anus, conjointly with extensive dilatations both of the vaginal veins and of those of the vesical plexus; in two instances, indeed, there were numerous varices of the extremely small twigs running beneath the mucous membrane at the neck of the bladder. When disturbance of greater moment results from dilatations of the vesical veins, a reduction of the hemorrhoids of the rectum ordinarily takes place, and, on anatomical examination, remnants are alone to be found.

In order that the anatomical relations just pointed out may be thoroughly comprehended, it will be desirable to acquire an accurate knowledge of the plexuses of the pelvic veins in their natural state. As the information on this subject to be derived from ordinary works is scanty and inadequate, I would recommend the study of Santorini's 'Observationes Anatomicæ' (Cap. de virorum naturalibus), as a useful guide. It is true that in the description of his labyrinth, and of his sinuses, he evidently often confounds diseased with normal conditions; the veins, however, about the prostate gland and the neck of the bladder are found to ramify in so peculiar a manner, even in healthy subjects, as almost to justify a belief that we have to deal with a portion of corpus spongiosum, enlarged in all its dimensions, although not confined and supported by any fibrous membrane. Reserving for a more fitting place a closer inquiry into these matters, I will here just briefly observe, that the vena dorsalis penis, and a few other branches which return the blood from the corpora spongiosa, rejoin Santorini's labyrinth in the vicinity of the prostate gland, and that from this labyrinth several very capacious but thinly coated sinuses emanate, and afterwards pass into the vesical plexus, or else discharge themselves through especial channels (as internal pudendæ) into the hypogastric vein.

These sinuses, together with their immediate continuations, frequently dilate in so extraordinary a manner, as to give to the prostate gland and to the inferior part of the neck of the bladder the appearance of being shrouded in a dense venous tunic. Their parietes become exceedingly attenuate, and no longer distinguishable without the aid of inflation, or of artificial injection. The external coat almost entirely disappears, shreds and little bundles of it only being now recognized at irregular points, where the caliber of the vein is tightly grasped and contracted. The valves are at the same time destroyed in the manner before indicated, so that the internal membrane is no longer smooth and even, but puckered,—wrinkled. In the superior branches of the vesical plexus there is dilatation, with thickening of the walls; the veins are stiff like arteries, and their parietes incapable of collapsing. Here likewise, however, the external membrane will give way at some points, and the internal coat protrude in pouch-like folds.

In these pouches, and in the prolongations of small venous twigs suddenly dilated, there occurs a very limited sub-inflammatory process, which seems to proceed almost imperceptibly, and gradually determines a coagulation of the blood in distinct concentric layers. Such coagula, for the most part spherical in shape, continue for a length of time subject to an interchange of action and reaction with the passing current of the blood, and there is deposited in them, under circumstances not yet well understood, phosphate of lime and magnesia, until the whole clot is transformed into a fossil mass, or *veinstone,* made up of concentric layers. When the dilated portion, or the protruding pouch, is thus entirely filled up, its walls commonly become atrophied; the inner surface of the vein assumes more of a cellular structure, and closes firmly round the veinstone, sometimes making it appear to be external to the vein. At other times the coagulation of the blood extends beyond the point where a veinstone has formed, the caliber of the vessel closes up, and an entire portion of the implicated vein is obliterated. In rarer instances veinstones occur free and moveable, within vessels which continue pervious to the current of the circulation. For the most part, however, the numerously formed coagula again liquefy, and there is no production of veinstones at all.

Much discrepancy of opinion has prevailed respecting the origin of these concretions. According to some, they form in peculiar sacs in the cellular tissue, external to the vein; according to others, they become deposited *between* the membranes, and ultimately get into the interior of the vein by means of a slow process of absorption. Otto, Tiedemann, Lobstein, Cruveilhier, Briquet, Carswell,[1] maintain that they form *originally within* the vein, and from numerous researches of my own, I am convinced that they are in the right. I have always found them connected with phlebectasis, and having repeatedly met with them in every phase of development, I consider myself warranted in affirming that they are engendered by a gradual, but direct deposition of calcareous matter from the blood within the layers of the coagula. On drying the coagula found within dilated veins, previously to their having coalesced with the internal membrane, they shrink together, grate under the knife, and exhibit calcareous induration at certain points—even in cases where it had not been at all suspected. On the other hand, veinstones, apparently quite hard, will likewise shrink, on desiccation, to very small bodies of irregular shape,—if the layers happen not to have received equable proportions of calcareous deposit. Those who content themselves with the examination of mature phlebolithes, can form no clear conception of their mode of development, because, at that advanced period the venous membranes in their immediate vicinity, and even beyond, have already suffered a material change. I may only mention, in conclusion, that the results of a chemical analysis, instituted by me in conjunction with Dr. Lehmann, entirely coincide with those obtained by Gmelin, who found phlebolithes to consist chiefly of phosphate, and some carbonate of lime, along with a portion of magnesia. The proportions of the organic constituents vary greatly, according to the period or stage of development, and to other contingencies. Detailed information on the subject of phlebolithes in general, and of their chemical relations in particular, is to be found in the elaborate treatise of Phœbus.[1]

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[1] Fasc. xi, vol. iii, fig. 1.

We must not suppose that phlebitis of the vessels connected with the bladder will always be so limited in degree, or stop short at products so unimportant as veinstones. Many an abscess of the cellular texture, within the perineum,—many a fistula,—may have a much closer affinity with an affection of this nature than has been hitherto suspected. I may affirm this confidently with reference to some diseases of the prostate gland, attended with suppuration. When it is remembered how exceedingly thin are the coats of the dilated vesical veins, and how slowly the blood circulates in them, it appears highly probable that such local suppuration may originate in phlebitis *with liquefaction of the venous coat,*—a process sufficiently rare in other veins.

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[1] De concrementis venarum osseis et calculosis. Berol. 1833.

Varices of the veins of the bladder immediately subjacent to, and causing an elevation of, the mucous membrane, are too rare to admit of a very faithful account being given of their development and their results. That so inconsiderable a number of observations of this description has been recorded,[1] is perhaps to be ascribed to the circumstance that, in examining dead bodies, the bladder is comparatively seldom taken out and minutely inspected; for I cannot attribute it to mere accident, that several examples of the kind have occurred within my individual experience. Two of these cases presented very inconsiderable varices, somewhat larger than a pin's head, which here and there appeared in groups in the unusually diffuse web of delicate vessels, not only at the neck, but likewise at the lateral surface of the bladder. Three cases exhibited round the commencement of the urethra, four or five venous pouches, rather larger than grains of hempseed, which reflected through the mucous membrane their purple colour interrupted, at intervals, by a thin layer of earthy incrustation.

It has been already stated that, in the female, phlebectasis is less prone to develop itself within the range of the bladder than in the vessels of the *vagina* and of the broad ligaments of the uterus. The disease here presents the same features as in the veins of the vesical plexus, except that no well-authenticated observation is recorded of venous tumours forming immediately beneath the mucous membrane. In the vaginal vessels the local peculiarities lead to the predominant occurrence of a form which recalls to mind the varices of the leg. We find, for instance, a vein dilated, perhaps, to the diameter of a goose-quill up to a certain distance: on, however, slitting it completely up, we search in vain at the extremity of the dilated portion for the prolongation of the vein, and are about to conclude that we have to deal with a cul-de-sac. Yet on persevering, we discover, considerably anterior to where the dilatation ends, the very minute orifice of the ulterior course of the vein, which, first making a lateral advance, presently dilates again, and finally returns in a parallel direction, and with a normal diameter. This condition extends far into the broad ligaments, but I have never met with it in any portion of the womb. Varices of the external labia pudendi are, however, not unfrequent, and death has been known to result from their rupture.

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[1] The examples cited from Morgagni, by Montégre, are not all genuine hemorrhoidal affections.

In other parts of the body, phlebectasis very rarely occurs, and the observations of the older authors on this point are to be received with due caution. Beneath the mucous membrane of the *œsophagus* I have repeatedly found varices concurrently with hemorrhoidal tumours at the anus—once in a very high state of development. Tumefaction of the veins of *the lips* is somewhat frequently observed at the point of reflection of the external skin. Varices likewise occur upon the *eyelids,*—a striking example of which was witnessed by Heidenreich.[1] Further instances will be found in some of the afore-cited works.

However important it might appear to study the changes in the other parts of the organism which seem to bear some morbific relation to phlebectasis, and more especially to hemorrhoids,—it seldom happens that subjects are examined who have died during the hemorrhoidal turgescence, at which period alone the reputed alterations in the liver and spleen could be properly estimated. The bodies in which I have myself found hemorrhoids, were of persons who had died of very various—for the most part chronic — disorders, so that even in the few in whom there happened, shortly before, to have been hemorrhoidal congestion, no sure inference could be drawn from the actual condition of the organs. Upon the whole, however, I have found—quite as often as diseased spleen and liver [2] — degenerations and tumours of various kinds within the abdomen, which, by pressing upon the veins, might well give rise to dilatations. In none of the cases was there any unusual dilatation in the system of the portal vein, or in the vena cava inferior and its larger branches. Vesal[1] and Berger,[2] however, found the hemorrhoidal vein dilated to the thickness of the thumb; Portal,[3] the vena portæ to the width of a portion of small intestine; and Rollin,[4] the ascending vena cava to that of the rectum, &c. In persons habitually affected with piles, dilatation of the right chambers of the heart is said to have been frequently observed; this, however, needs confirmation.

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[1] Schott (Wurtemb. Corr. Bl., vol. viii, No. 38) saw death ensue upon their rupture.

[2] The observations made by C. H. Schultz on the development of hemorrhoids (Hufeland's Journ., Mai 1837,) albeit hypothetical, are not devoid of interest. He says: "The blood-vesicles of the portal system having here attained their last stage of vital activity, to be finally expended in the liver for the secretion of bile, sink, —when the latter secretion stagnates and the portal circulation is consequently arrested,—to the roots of the veins and there give rise in a manner partly mechanical, partly dynamical, to the development of varices and hemorrhoids. The probability

Concerning the peculiar relation recognized by most surgeons between fistula ani (which is generally of hemorrhoidal origin) and pulmonary phthisis, I have no precise or satisfactory explanation to offer. Bushe[5] says, that the constantly recurring impulse of the cough irritates and dilates the veins, to which the cellular texture, owing to the absorption of the fat, can no longer afford adequate support, and that the dilatation is augmented by consecutive stagnation in the portal system, arising from the hurried character of the pulmonary circulation. It is more probable, however, that, in persons who die of phthisis shortly after the healing of fistula ani, tubercles preexist in the lungs,—remain latent so long as the derivative suppuration lasts,—but soften down so soon as it is checked.

It is obvious, from what has been premised, that the numerous diseases which are referred to suppressed hemorrhoids, namely, apoplexy, chronic bronchitis, gout, lithiasis, &c., cannot, with *anatomical precision*, be traced to that source.

In concluding this article, we ought not to overlook the fact, that dilatations of the veins may, when associated with certain peculiarities of constitution, easily lead to formations belonging to the class of fungus hæmatodes or of erectile tumours, and eventually to destruction through cancerous softening. An example of malignant tumour from varices of

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of such a result is increased by the circumstance of the vesicles of the portal blood being of greater, its fluid parts of less,—specific gravity than in any other set of vessels.

[1] De Hum. Corp. Fabr., l. v, cap. 15.

[2] Diss. de Hemorrhoid, ultra modum prof.

[3] Lobstein, vol. ii, p. 602.

[4] Journ. de Méd. de Roux, &c., vol. xxxii, p. 44.

[5] Diseases of the Rectum.

the leg is related by Warren,[1] and Cruveilhier's cases[2] of erectile tumours on the upper extremities are of a similar character (see Arteriectasis), the cutaneous, the muscular, and even the nervous textures having here and there been found degenerated into grape-like varicose tumours.

The forms of phlebectasis hitherto considered are perfectly distinct from certain other dilatations, some of which are of a physiological nature, such as those of the spermatic and uterine veins in pregnancy; others supplemental to the circulation, when interrupted by the closure of large venous trunks; others consequent upon the development of vascular tumours, in various organs,—the thyroid gland, for instance; whilst finally others again appear to result from dilatation of the right side of the heart, and stagnation within the range of the lesser circulation.[3] In conclusion, we may direct attention to those cases in which the umbilical vein remains patent from the time of birth, or becomes so in consequence of external injury. Here the subcutaneous veins of the abdomen are wont to dilate, and that sometimes to a very considerable degree.[4]

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[1] L. c., p. 453.

[2] Livr. 23, pl. iii, iv, and livr. 30, pl. v.

[3] Cases by Cassau (Arch. Gén., 1827, vol. xiii, p. 77,) and Girgensohn (Mittheil. Rigaischer Aerzte, 1839, vol. i, p. 18,) where, simultaneously with dilatation of the right cavities of the heart and of the vena cava, the coronary arteries of the heart were widened to the caliber of the subclavian vein. Lobstein (vol. ii, p. 602.), dilatation of the ascending vena cava, completely displacing the heart; taken from the Misc. Nat. Cur., &c.

[4] See Cruveilhier, liv. xvi.

# CHAPTER III.

## DISEASES OF ARTERIES.

### SECT. I.—ARTERITIS. INFLAMMATION OF ARTERIES.

NOTWITHSTANDING a great *general* analogy between the inflammatory processes in the several systems of the circulating apparatus, certain distinctions are nevertheless apparent, according as the disease has its seat in the centrifugal or the centripetal vessels, or in the great central organ itself. The following sketch will therefore, on the one hand, gain in conciseness by a frequent reference to the chapter on phlebitis and the section on endocarditis, whilst, on the other, certain characteristic features peculiar to arteritis, and no less physiologically true than practically important, will have to be considered more in detail.

The disease is very rare, and for the most part developed under very different circumstances than phlebitis. It is usually coincident with, or consecutive upon the inflammation of other organs; and most frequently originates from traumatic causes. Arteritis is sometimes a sequence of the retrocedence of exanthematous diseases (Portal), and is sometimes a consequence of rheumatic affections. It has been divided, first, into *general arteritis,* believed to invade a large portion, if not the whole, of the arterial system, and into *partial arteritis,* confined to individual trunks and branches; secondly, into *acute* and *chronic.* I confess never to have myself seen a case of general inflammation of the arteries in the sense of the above distinction, and I am less disposed, even in the examples frequently cited by P. Frank, to recognize an inflammatory affection of the arterial coats, than a peculiar—very possibly inflammatory—alteration of the blood, causing the inner surface of the vessel to become tinged, probably after death. In the description of endocarditis, allusion will be made to the difficulty of laying down sure criteria for a correct estimate of the redness found within blood-vessels on dissection; and several circumstances will there be specified, under which the internal membrane of the vessel, without being inflamed, exhibits various morbid tints. What is there said need not here be anticipated; it may, however, be stated generally, that there are many other instances besides those adduced, in which such a dye indicates nothing beyond imbibition.

We have seen that the veins may, by slight irritation of their internal membrane, be excited to inflammation: this is not the case with arteries. Trousseau and Rigot[1] found that neither alcohol (of 36 degrees; sp. gr. 0·835), nor dilute nitric acid, nor putrefying animal substances, determined any inflammatory reaction in their internal membrane; such a result being only attainable by acrid substances, like the tincture of cantharides and of euphorbium, employed by Sasse.[2] This may be accounted for by the different structural relations of arteries and veins; in the latter every irritant quickly penetrates the internal membrane, and the peculiar, but very thin, felt-like fibres of the so-called middle tunic, so as to reach the cellular coat; in the latter, there is interposed between the internal non-vascular membrane and the cellular coat (rich in blood-vessels and nerves), a pretty strong layer of elastic texture, possessing a very low degree of vitality, so that the cellular coat is endued with all the vegetative and animal activity of the artery.[3] Accordingly, this latter coat is highly susceptible of every irritation, and readily inflames when acted upon by either chemical, dynamical, or mechanical influences.[1]

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[1] Arch. Gén., 1826 and 1827.

[2] Diss. de vasis Infl., Hal., 1797.

[3] Hence the necessity, in tying arteries, where it is important to produce vigorous adhesive inflammation within the shortest possible time, of drawing the thread tightly together, so as to divide and sufficiently irritate the internal and middle membranes, and thereby speedily establish a plastic plug which shall adhere firmly to the parietes of the vessel before suppuration commences in the cellular sheath. Subsequent interference should be abstained from, lest the suppuration extend beyond what is absolutely requisite. For this very reason, the tying of a wounded and already inflamed artery is of doubtful issue, and the use of *ligatures d'attente*, of *Presse-artères*, and all such contrivances, is to be condemned as retarding or frustrating the main object of ligature, namely, adhesive inflammation. For when the external tunic is thus largely exposed and permanently irritated, the inflammation goes on increasing, matter forms which leads to destruction of the plastic product, together with the adjacent membranes, and eventually to fatal hemorrhage.

With such important differences in the anatomical and physiological relations of the several arterial membranes, the process of inflammation must necessarily manifest itself in a very different manner in each of them. First, as regards inflammatory redness, we observe in the internal membrane a uniform faint hue of a dingy reddish-brown, which darkens by degrees, and most probably results from the action of the blood, modified through inflammation of the arterial parietes. The external membrane, on the other hand, reddens from copious injection of its delicate vascular tissue.[2] The elastic membrane at first gives little or no evidence of having suffered any change. At a more advanced period, the cellular sheath of the artery swells, serous infiltration ensues, not only betwixt the elastic fibres, but also in the cellular tissue connecting the respective membranes, and surrounding the arterial tube. In this condition the several membranes readily separate from one another, are softer and less elastic than natural, and easily tear. The internal membrane has lost its lustre, and assumed a darker coloration. Presently it is covered with a layer of plastic lymph, (partly by coagulation of the fibrin in the blood, partly by exudation through the parietes of the vessel,) which goes on accumulating, in concentric layers, until a plug is formed, whereby the caliber of smaller arteries is thoroughly closed. This I have met with in two instances of acute idiopathic arteritis; one in the common iliac, the other in the femoral artery. In the aorta things do not generally appear to go so far; when inflamed, a few firmly adherent layers only of false membrane form upon its internal coat.[1]

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[1] The recent researches of Henle (Allgemeine Anatomie, p. 494,) establish for the arteries six distinct membranous layers: 1st, a delicate pavemental epithelium; 2dly, a very delicate membrane with reticulate fibres; 3dly, a membrane with longitudinal; 4thly, one with circular fibres (the elastic membrane); 5thly, a delicate membrane, consisting of proper elastic fibres—this is present in the large arteries only; 6thly, and lastly, the cellular sheath, the only membrane provided with blood-vessels. The rare appearance of the different forms of inflammatory exudation on the inner surface of the arteries, when not denuded of the internal layers, is explained, if not confirmed, by this minute account of the structure of the arterial coats.

[2] Spangenberg (Horn's Archiv, vol. v, fasc. 2, 1804,) mentions an arborescent reddening of the internal membrane. I have, however, never myself observed the development of vessels in this membrane in the first instance, though, at a late period, when obliteration is about to ensue, it may become conspicuous. Of course, hæmostatic repletion of the vasa vasorum is not to be confounded with such inflammatory injection.

Some writers have maintained that it is often impossible to detect the exuded matter in inflamed arteries, because it is carried away with the stream of blood. The question then arises, what becomes of it? unless it be assumed, that it directly returns to the liquid state. Otherwise it would (just as has been shown in the case of phlebitis) necessarily induce changes within the capillary system, or, if it contrived to get beyond the latter, effect a coagulation of the blood at some ulterior point. Sometimes this last event seems really to take place; thus, in the instance of inflammation of the femoral artery just alluded to, I detected coagulation of the blood, and incipient inflammatory plugging in the femoral and also in the left jugular vein. In Hodgson's case[2] there arose, during the course of an inflammation of the arteries of the right lower extremity, a similar one in the arteries of the right arm. In rare instances plastic exudations very rapidly form within the track of the capillary system of the inflamed artery, just as in endocarditis (vide article); they evince, however, less proneness to suppuration than the lobular inflammations consequent upon phlebitis. Thus, in a case of inflammatory exudation occupying the surface of an atheromatous ulcer at the arch of the aorta, I found isolated portions of the spleen densely infiltrated with a yellowish brown matter, and wedge-shaped portions of the cortical substance in the left kidney of a dirty yellow colour, and pervaded with exuded fibrin. If the cases related by Bizot[3] were in reality inflammation of arteries, the more or less general anasarca which accompanied them should be reckoned amongst the secondary phenomena of arteritis. But local irritation, competent to engender inflammatory products, will always leave vestiges cognizable after death; which event, in inflammatory diseases, is immediately owing to some of those products being—either *absolutely,*—or *relatively* to the actual condition of the vital powers—insurmountable.

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[1] Compare the observations of Spangenberg (l. c.); of Bouillaud (cited by Andral and Lobstein); of Farre (in Hodgson); of Thierfelder (Ammon's Monatsschrift, vol. iii, fasc. 1 and 2, 1840.)

[2] L. c.

[3] Mém. de la Soc. de Méd. Obs. vol. i. Compare note in the section on the formation of semicartilaginous patches in the arteries.

It is remarkable that the secondary symptoms, which play so decided a part in phlebitis, are very seldom observed to follow arteritis. This cannot be attributable to death ensuing earlier in the latter than in the former disease; for in phlebitis lobular abscesses frequently form with great rapidity, whilst in some cases of arteritis the fatal issue is deferred until a very late period. The real cause seems to depend upon some peculiarity in the arteries, in virtue of which plastic matter only is deposited within their canals, and inflammation restricted to the adhesive form. Hence, the more solid product of morbid exudation is not under the control of the heart's impulse, and can alone pass into the circulation when the exudation and the coagulated blood, or the fibrin deposited by the blood, are too scanty to close up the arterial caliber completely, or for a sufficient length of time. This peculiarity manifests itself most distinctly in the tying of arteries, after which operation, notwithstanding the long sojourn of a foreign body, there results a firm plug and adhesion, unless from a concurrence of extraordinary circumstances, diffuse suppuration is induced, which then commences in the surrounding parts and in the cellular tunic, and only *indirectly* invades the lining membrane of the artery,—the plug being originally *always* of a purely plastic nature. In simple arteritis the circulation in the involved artery is, in like manner, mechanically arrested, up to the nearest diverging branch, by the product of the inflammation, just as it is from the application of a ligature. Should suppuration ultimately occur even *under these circumstances,* it remains purely local,—shut out from the general current of the circulation, however far it may extend downwards in the direction opposed to that of the heart. Should the purulent softening extend upwards, so as to destroy the closing plug, the blood-stream will find its way back to the inflamed portion. Here, however, the artery is no longer entire, since the suppuration usually begins in the external coat, and in its progress destroys the others. Thus the blood, meeting with no resistance, perforates to a greater or smaller extent the softened membranes. If this happens with an artery to which a ligature had been attached, a fatal hemorrhage necessarily ensues, unless a fresh ligature be instantaneously applied higher up. In spontaneous arteritis occurring in parts previously sound, there ensues either extravasation, or a so-called *diffuse spurious aneurism,* the hemorrhage being restrained by the thickened and indurated cellular texture in the neighbourhood of the ruptured part. Such occurrences sometimes follow external injury; but in spontaneous arteritis they are so rare, that I can call to mind but a single authentic example, namely, the case before cited from Hodgson's work.

The only secondary affection of proportionate frequency in arteritis is what is termed *spontaneous gangrene,* long known by the appellation of gangræna senilis; a name now obsolete, in consequence of its having been satisfactorily proved that the malady may affect any age, not excepting childhood.[1] Dupuytren [2] was the first who ascribed spontaneous gangrene to arterial inflammation; and Cruveilhier[3] upheld this view, partly by new observations, partly by experiments upon animals, having succeeded in determining gangrene artificially by injecting irritant fluids into the arteries of the extremities. He showed a complete occlusion, not only of an arterial trunk, but likewise of its ramifications (restraining all afflux of blood whether direct or collateral, to the respective part) to be indispensable to the establishing of gangrene. A closing up of arteries like this need not necessarily originate in an ordinary acute inflammation; any cause, as Carswell has demonstrated, sufficing, provided it produce a coagulation of the blood within those vessels,—for example, irritating arterial membranes when extensively ossified. Ossification of the arteries would not, therefore, as was formerly believed, appear to be the proximate, but rather the accidental, remote cause of spontaneous gangrene.

Inflammation sometimes gives rise, at certain points only, to purulent formation between the membranes of the larger arteries. Andral mentions a case in which some half dozen of little abscesses were formed beneath the inner membrane of the aorta; Lobstein saw the same thing in an aneurismatic aorta. Bizot questions the accuracy of these observations, believing softened atheromatous deposits to have been mistaken for abscesses; it is, however, hardly to be suspected that men so cautious and experienced should have committed such an error. These abscesses are no doubt very rare phenomena; they occur between the cellular sheath and the middle layers, and are seldom extensive, probably never encircling the whole of the arterial cylinder. From the accumulation of pus, the middle layers soften and liquefy; the pus ultimately making its way into the caliber of the artery. The cellular coat firmly adheres to the adjunct parts, and the ultimate consequence is an aneurismal dilatation, for the most part not very extensive, at the seat of the abscess. This dilatation is caused by the imperfect reproduction of the middle and internal layers, and the consequently diminished resistance to the pressure of the blood. I have had an opportunity of tracing this whole procedure with some degree of precision in the common iliac artery. Finally, the smallest twigs of the pulmonary artery in the immediate vicinity of tubercular cavities, and of lobular abscesses, are found filled with pus, which may, however, have gained entrance otherwise than by inflammation of their membranes.

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[1] See Medical Gazette, April and June 1839.

[2] Leçons Orales, vol. iii, p. 268.

[3] Livr. 27.

In the great majority of cases the inflammation of arteries is of a purely adhesive character, and terminates in obliteration. The process whereby the latter is accomplished, has been elucidated through the introduction of ligature. It has been attentively investigated by Jones[1] through its various stages, with due reference to most of the concomitant circumstances. A still clearer insight has however been afforded by the experiments of Stilling.[2] These relate to the healing of wounded or tied arteries in animals; and we may draw, from a comparison of their results with the analogical process of adhesion and closure in the fœtal vessels, a tolerably fair inference as to the gradual accomplishment of obliteration in spontaneous arteritis.

When an artery is divided, the hemorrhage, apart from surgical aid, is in the first instance restrained by the retraction of the vessel and the contraction of its mouth, as also by the external and internal clot, and afterwards more effectually by plastic exudation from the lips of the wound. At a later period this exudation on the one hand, and the plug formed within the vessel on the other, complete the healing and obliteration of the wounded artery. The first of these acts of course cannot apply to spontaneous arteritis; here, however, the plastic matter which is thrown out into the neighbouring cellular tissue and betwixt the membranes of inflamed veins and arteries, becomes highly important, from its promoting the development of the vasa vasorum and the creation of the new vessels requisite for the adhesive process. The influence, however, of the plug forming within the arterial canal is very considerable; for through its plasticity alone a stagnation of the circulating current is effected, without the aid of any mechanical support, like that afforded in the case of divided arteries or of those tied in their continuity.

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[1] Jones, J. F. T. A Treatise on the Process employed by Nature in suppressing Hemorrhages, &c. Lond. 1805.

[2] Die Bildung und Metamorphose des Blutpfropfes oder Thrombus (the development and transformation of the blood plug or thrombus,) &c. 1834. In this work Stilling communicates seventy experiments on animals—no less carefully performed than accurately described.

Let us now proceed to study the formation and transitions of a thrombus conformably to the researches of Stilling, bearing in mind, that the process will vary in individual cases according as a greater or minor degree of inflammation renders the plug more or less rich in plastic material. Immediately after the tying of an artery, there ensues stagnation of the blood to the next great collateral branch, with accumulation of blood-globules, first of all near the ligature, while coagulation of the fibrin from the fluid constituents of the blood, follows by and by. For this reason the thrombus is, during the first eighteen hours, rather of a conical figure, and somewhat deeply tinged at its base; while the apex is of a yellowish colour, almost like that of an inflammatory crust. Upon the whole it is of very slender cohesion, is attached merely at the base, and that loosely. All the interstices, however, progressively fill up; partly through continuous coagulation from the blood, partly through plastic effusion from the parietes of the vessel. The plug now approaches more nearly to a spindle shape, having a short obtuse base resting upon the part where the ligature was applied; a thick body; and a longer tapering portion, or apex, pointing towards the heart. Its colour is uniformly pink or brownish red; and its consistency at this period somewhat firmer at its periphery than at its centre. It now begins to attach itself to the contiguous parts, so that on being removed, delicate filamentous adhesions are observed to connect it with the parietes of the vessel; its apex however still remains free. Where there has been much and repeated exudation, the plug exhibits externally several concentric layers. I have frequently found it thus after ligature. Stilling once met with two plugs lying side by side, and dovetailing with each other, so to speak, like two kernels within one nutshell. So soon as the plug is completely formed, the plastic exudation terminated, and the inflammatory process has ceased to alter its own products, the organism strives to appropriate to itself these new formations. This attempt is, however, not altogether successful; for, whilst the plastic matter assumes a vegetative vitality perfectly identical with that of other parts of the body, most of the constituents of the thrombus undergo a process of removal. There is, however, this peculiarity, that, unlike perfectly foreign bodies, this thrombus is not expelled by suppuration or isolated by capsular involution, but enters into an immediate connexion with the vascular system, and passes through certain transformations which end in its entire resolution. The walls of the vessel, namely, coalesce with the thickened adjunct cellular tissue, or with the exudation from the wound, afterwards tumefy, and gradually drop all distinction of their several membranes, so that at length the cellular tissue with its plastic infiltration, the arterial walls, and the thin layer of exudation encircling the thrombus to its very apex, are consolidated into one mass. Throughout this mass, which is white and of a sero-lardaceous character, there ramifies a network of very delicate vessels, which densely surrounds the firmly adherent thrombus.—In injected preparations Stilling could plainly distinguish the mass just described from the now vividly reddened thrombus; the latter was likewise permeated by the injection, and so equably, as to obviate all question of extravasation. The periphery was much softer than the centre, and more densely pervaded by the injecting substance.—Afterwards the plug becomes less and less consistent,—porous, so to speak; its proper substance nevertheless is firmer than before, receives more of the injecting mass, which is confined within smooth tubular canals, wide enough, in some instances, to admit a human hair; and yet it appears that a direct transition of these tubular canals into the close network of vessels in the plastic mass can nowhere be distinctly traced.[1] — Stilling likens the structure of the thrombus at this period to that of the placenta and of the corpora spongiosa.—From this time the thrombus, engaged in the process of liquefaction, is rapidly dispersed; the parietes of the artery gradually collapse, and finally cohere, and that generally for some distance beyond the point to which the plug and the plastic exudation had extended. This is seen in small vessels after from twenty to twenty-five days, but in large ones not until after four or five weeks.

If the artery on closing up preserves its continuity, the obliterated patch serves, as a thick cellulo-fibrous cord, to unite its two extremities, and as soon as the collateral circulation has become established, its peripheral[2] or inferior portion is again pervious to the circulating current. If, however, the vessel has been divided between two ligatures, or if at both divided extremities, torsion has been practised, the interval between the two free portions of the artery will have become considerable, and the obliterated extremities very difficult to recognize in the scar-like condensation of the newly-formed cellular tissue. The ligatures,—which at the moment of application had divided the internal and middle membranes of the vessel,—sever the external membrane through suppuration, during the proceedings above described, and, after a shorter or longer interval, come away.

A very remarkable vascular formation has, in many instances, been observed in the middle of the thrombus and of the obliterated arterial cord. Namely, besides the irregular cells and canals whereby the thrombus is, so to speak, excavated and dispersed, Stilling discovered in several of the preparations a thoroughly organized vascular canal traversing tortuously the centre of the plug. It appeared to originate directly from the still open portion of the arterial trunk, and in one case, distinctly branched off into two. In like manner, Lobstein[1] met with an arterial vessel of the caliber of the stylo-mastoid artery, running lengthwise through a femoral artery, obliterated two years previously by tying; and Blandin,[2] a similar vessel in the centre of a fibrinous coagulum, filling up a femoral artery upon which a ligature had been placed eight years before,—the vessel was very plexiform at the extremity of the obliterated portion. To these may be annexed Barth's case,[3] in which a central canal passed through the middle of an old plug filling up the abdominal aorta. Comparing these observations with analogous examples in the venous system, (vide Phlebitis,) we would recognize in them nature's efforts to restore the circulation through the closed artery; a view rendered the more plausible from the fact that Stilling and others[4] have, after the complete obliteration of tied arteries, repeatedly witnessed a number of new vessels shooting forth in arborescent distribution from the stump.

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[1] It is to be regretted that the composition of the injecting mass is not given, and that the microscope was not made use of in these examinations.

[2] The process in the peripheral and in the proximal extremity of the obliterated artery is not materially different. Upon the whole, the transformation of the thrombus is more rapid in the former. (See Stilling's work.)

I am unacquainted with a single example of the idiopathic occurrence of *chronic arteritis*. Most pathologists attribute the formation of the semi-cartilaginous patches, the ossifications, the ulcers, and the true aneurisms of arteries to inflammation; it will be shown, however, in the proper place, that the inflammatory process does not stand in any causal relation to these affections. It is not, however, to be denied, that extensive ossification or atheromatous degeneration may *call forth* a limited degree of chronic inflammation in the arterial membranes. I have myself met with several examples of this kind; indeed, the softening of atheromatous deposits is wont to ally itself with irritation, and with increased fulness of the blood-vessels in the cellular membrane, although without warranting us in considering such concomitants, any more than chronic inflammation itself, as the actual exciting cause.

The organic changes following arteritis of this description frequently consist in a diseased condition of the arterial membranes, in the immediate neighbourhood of aneurismal sacs, or where numerous ossific patches happen to irritate or tear the internal membrane, or lastly, in the vicinity of arterial ulcers. The alterations manifest themselves in a deep, dingy, brownish red colour extending to the middle tunic, in a densely injected state of the vasa vasorum of the cellular sheath, and in a deposition of plastic material within the caliber of the vessel. In the above situations, these fibrinous deposits accumulate in masses which adhere firmly to the arterial parietes, and are made up of single layers arranged in an imbricated manner; in one instance, I found them so thickly congregated at the arch of the aorta as to have blocked up the left subclavian artery. This state of things, by no means rare, has been well figured by Carswell.[1] Where death has been preceded by a protracted struggle, and the circulation thus made to stagnate very gradually, these deposits increase still further, owing to the mechanical impediment they offer to the circulating current, and it then becomes easy to distinguish the older layers from those last formed.

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[1] Anat. Path., vol. i, p. 298, par. 334.

[2] Journ. Hebd., Mai 1840.

[3] Presse Méd., 1837, p. 62.

[4] Thus, Von Ebel (De Naturâ Medicatrici sicubi arteriæ vulneratæ et ligatæ fuerint. Giessen, 1826.) Manec (Traité de la Ligature des Artères.)

## SECT. II.—STOPPAGE, STRICTURE, AND CLOSURE OF ARTERIES.[2]

In the preceding chapter we have considered the stoppage of arteries by an inflammatory product, which process, particularly as resulting from adhesive inflammation designedly called forth, we have traced step by step to the final closure of the vessel. The organism tolerates such an interruption to the circulation with astonishing ease,—the connexions of the small arterial branches above and below the point of obliteration dilating pretty rapidly, and thus establishing a collateral circulation, equal to the necessities of the parts concerned.—In this manner, a complete closure, even in the thoracic aorta, may prove harmless, provided the obliteration is of limited extent, and of gradual formation, allowing the collateral vessels time to dilate proportionately to the great demand made upon the circulation by the lower half of the body. Thus the sudden *tying of the aorta* does not (as is shown by Astley Cooper's[1] experiments on dogs) necessarily occasion death.[2] The anastomoses of the lumbar and mesenteric arteries effectuate the further course of the blood,—so readily, indeed, that on tying the aorta above the cæliac artery in the dead body, a thin injecting fluid thrown into the ascending aorta finds its way into the arteries of the lower extremities. Nevertheless, A. Cooper's application of a ligature to the abdominal aorta, during life, proved unsuccessful. The slower obliteration of the aorta by spontaneous inflammation is not directly fatal, but is productive of abiding and very material disturbance to the health, owing probably to a large extent, not of the trunk alone, but likewise of the proximate branches becoming impervious.[3] Death ensues ere very long, and is sometimes, where the plug extends far into the smaller branches, immediately preceded by gangrene of the lower extremities.[4]

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[1] Fasc. vii, pl. iii, fig. 2.

[2] Under this head will be considered the results not only of inflammation and other diseases, but also of imperfect development.

Closure of the arteries of the head is likewise easily borne, as is amply attested by surgical experience. Thus Kuhl tied both common carotids, within a short interval of each other, with the most favorable result.[5] In animals, where the cerebral vitality is not so important as in man, the application of ligature to both carotid and both vertebral arteries simultaneously, is not necessarily fatal, as shown by the experiment of Sir A. Cooper upon a dog.[5]

The human organism, would, indeed, be unable to resist a sudden shock of this kind. On the other hand, the great trunks of the extremities have in numerous instances been tied,—life being preserved, and the limbs recovering all their vigour.[1] Every surgeon is familiar with instances of the closure of small arteries caused by spontaneous inflammation, the pressure of tumours, &c., or by artificial means.

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[1] Med. Chir. Transact., vol. ii.

[2] Compare Pirogoff, Annal. der Chir. Klinik zu Dorpat, 1839, p. 219.

[3] As, probably, in Schlesinger's case, (Casper's Wochenschrift. 1836,) in which the artery was obliterated at the point where it penetrates the diaphragm; and in Barth's case, where it was obliterated from the inferior mesenteric downwards. (Barth. Arch. gén. 1835, and in the 'Mém. sur les retrécissem. et les oblitérat. spontanées de l'aorte' in the Presse Médicale, 1837, Nos. 58-66, where almost every recorded example is to be found.)

[4] See Barth's seventh case, in which fibrinous obstruction was brought on by aneurism of the aorta.

[5] Medical Gazette, 1835. Guy's Hosp. Rep. vol. i, p. 457.

The reestablishment of the circulation subsequent to ligature, &c., is principally effected by dilatation of the free collateral branches, although different processes have been at times observed. Thus, as the result of experiments upon animals, F. J. C. Mayer has described and figured two curved lateral vessels, doubling, as it were, the point of the ligature, and uniting the two extremities of the carotid. Reference has already been made to the arterial shoots which Stilling saw growing out of the stump of the vessel and to the central canal, found traversing the thrombus. In general, however, the detailed account given by Hodgson appears to be substantiated. Immediately after the tying of a main artery, the circulation is carried on through the innumerable small ramifications; a few of these afterwards dilate considerably more than the remainder of the anastomoses, which latter soon resume their original caliber. It is remarkable, that in general the twigs alone dilate, whilst the trunks of the branches from which they emanate are, even after a lengthened period, hardly at all enlarged. To this, however, the collateral circulation, in the case of obliteration of the aorta, is an exception. Hence it is obvious how greatly the development of a collateral circulation may become impeded by pressure arising from the habitual posture of a patient; from tightly bandaging the affected part; from division, by a broad transverse wound, of a majority of the lateral branches; from compression and ultimate obliteration of the latter by tumours; or, finally, from the circulation becoming enfeebled through loss of blood or lowering treatment.

Atheromatous changes are commonly productive of dilatation; sometimes, however, the deposition within a given compass is so copious as to constrict, if not close the vessel.[1] This is a consequence, in particular, of excessive deposition of calcareous products between the walls of an artery, a process sometimes associated with inflammation which leads to the formation of an adhesive plug and eventually to obliteration. *Atrophy* of the part involved is the common result. I have repeatedly witnessed instances of this in the uterus of elderly women; and Carswell has figured it in both uterus and spleen.[2] A coarctation of arteries so originating ought not to be confounded with that *consequent upon* the atrophy of parts they supply. Otto,[3] for example, remarks that the arteries of members which have been long palsied, or of organs that are worn out, diminish in size; Carswell,[4] again, has represented a kidney atrophied by hydatid degeneration, the artery of which appears very considerably contracted, whilst the vein retains its natural caliber.

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[1] See Hodgson (l. c.); Sir A. Cooper's examination of the parts where ligature had been applied to the external iliac eighteen years previously (Guy's Hosp. Rep. vol. i, p. 43); Key's,—twelve years after ligature of the subclavian vein (ibid. p. 59), with figures; and Saloman's,—one year after ligature of the common iliac (Fricke's Zeitschrft, vol. xiv, fasc. 1.)

But even where the obliteration of arteries proceeds from a degeneration, of their membranes, nature, of its own accord, establishes a collateral circulation. This is proved, not only by the cases collected by Hodgson, of the spontaneous cure of aneurismal tumours, through closure of the affected artery, but also by several of the examples adduced by Barth, of contraction and closure of the aorta at certain points, through ossification. Of the latter, the most remarkable is that recorded by Goodisson, of a woman in whom the aorta from the inferior mesenteric downwards, the left iliac throughout, and the right iliac for one half of its course were found impervious,—and where, nevertheless, a collateral circulation had been maintained through the intercostal and lumbar, and through the internal mammary and the spermatic arteries. The ossification in the aorta had the appearance of a hollow inverted cone, whose interstices were filled with a solid organized plug, resembling the fleshy stomach of birds. The affected arteries were surrounded outwardly with a gelatino-cartilaginous substance. In the body of a diabetic patient, Hodgson found one renal artery thoroughly choked up by an atheromatous and calcareous mass,[5]—the kidney, nourished by the lumbar artery, and the vessels of the renal capsule being found healthy. In another case,[1] the cœliac artery was completely stopped up by the pressure of an aneurism, as was the superior mesenteric by ossification, without detriment to the organs concerned. Numerous instances of the kind might be quoted, but I will merely refer to that of Jadelot,[2] where both carotids were found closed through ossification.

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[1] See Barth and Otto.

[2] Fasc. x, pl. iii, figs. 4, 5.

[3] Path. Anat. par. 192.

[4] Loc. cit. pl. i, fig. 5.

[5] Hodgson.

Other growths and tumours developed betwixt the membranes of the vessels may determine, in like manner, the contraction and adhesion of arteries.[3] In some rare instances of confirmed cancerous habit, the great arteries, including the aorta, are found obstructed with masses of clotted blood mingled with cancerous substance.[4]

It will be shown in the proper place, that hypertrophy of the left ventricle of the heart in all probability sometimes depends upon *congenital narrowness of the aorta,* at its origin, and of a large proportion of the arterial vessels; some remarkable cases in point have been brought forward by Meckel the elder, and by Andral. Congenital narrowness of the pulmonary artery will be treated of under the head of *morbus cæruleus.* There are, besides, instances of coarctation and complete closure of the aorta decidedly originating in arrest of development. Such examples are, however, far from common. I am acquainted with seventeen, nine of which have been collected by Barth,[5] the tenth in which the aorta had but two semi-lunar valves, is described and figured by Otto,[6] and the eleventh by Römer.[7]

*Stricture* or *closure* of the vessel occurs either at the mouth of or somewhat below Botalli's duct, the coats of the aorta manifesting no signs of disease. The constriction is mostly confined to one point, like that produced by ligature, the tube there contracting to the caliber of a goose or crow quill, and presenting a circular or irregularly cleft shape. In Legrand's case, there was a double stricture, one of a slight character at the mouth of Botalli's duct, the other more marked, eight lines below; in the latter there was a diaphragmatic septum with a roundish orifice, a line and a half wide. Above, the aorta was always irregularly, often very considerably dilated; below, it generally had its natural width, and was, along with its branches, in a few instances only, somewhat narrower than usual. In two individuals the lower extremities fell short of their due proportions, and showed during life a constant deficiency of physical power. The circulation was (as is detailed in most of the cases) carried on by means of a complicated network of dilated and very tortuous branches of the first intercostal, the internal mammary, the subscapular, the thoracic, and the rest of the intercostal arteries, together with the epigastric. Botalli's duct was found closed (except in two instances, where a slender probe could be passed through it); whence Meckel concludes that the aortal coarctation could not have taken place before or even shortly after birth, otherwise the duct would have remained open. The heart was for the most part hypertrophied and dilated, particularly its left ventricle. In four individuals death ensued from rupture,—once of the right auricle, once of the right ventricle, and twice of the dilated ascending aorta. In several instances dropsical accumulations were observed for a longer or shorter period before decease. This kind of stricture or obliteration of the aorta occurred in individuals of various ages, from that of seven to that of ninety-two years; thirteen were males, and four females; several of whom enjoyed good health to the last, while others previously suffered from a variety of ailments of the respiratory and circulating functions, referrible, in a great measure, to hypertrophy of the heart. Two died of tubercular affection of the lungs. In several, the above diseased condition of the aorta was accidentally discovered during dissection.

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[1] Hodgson.

[2] Description of a preternatural human head, &c.

[3] Stenzel Dissert. de Steatomate Aortæ. Viteb. 1723. J. Frank, t. viii, p. 329.

[4] See cases by Velpeau, Nicod, and Churchill, in Barth and Otto.

[5] Loc. cit.

[6] Seltene Beobachtungen (rare cases), ii, p. 66.

[7] Römer, Oesterr. Jahrbücher, vol. xx, fasc. ii.

### SECT. III.—FORMATION OF SEMICARTILAGINOUS PATCHES AND OF ATHEROMATOUS DEPOSITS UPON AND BETWIXT THE ARTERIAL MEMBRANES.

These two morbid changes being for the most part associated with each other, it will be found expedient to consider them both under one head. In persons who have passed the meridian of life, a cadaveric examination is rarely made without some trace of them being discovered. Yet their pathological import has been little attended to, nay, from their probably originating without obvious symptoms, no attempt has been made to connect them with any distinct morbid process. To accomplish this, it would be necessary on every occasion to examine the larger arteries, devoting particular attention to those cases in which the morbid product is unusually abundant, or occurs at a period of life ordinarily exempt from the disease, and carefully ascertaining the coincident changes in other organs. Mere anatomical description will amply show how much both those arterial diseases must influence the whole circulating system, along with its subordinate functions.

*Semicartilaginous patches* occur throughout the whole of the arterial system on the free surface of the internal membrane. They, in all probability, originate from a semi-fluid, almost gelatinous-substance, of a pale yellowish or reddish colour, forming a layer of greater or less extent upon the inner surface of the artery. In the aorta and larger vessels this layer is mostly scanty, but in the smaller vessels often more abundant. Thus Bizot[1] found a considerable portion of the tibialis antica almost replete with it. It is doubtful whether the layer be the result of morbid exudation from the internal membrane, or be deposited immediately from the blood. The latter assumption appears more satisfactory, if it be considered that neither the internal nor the other arterial coats suffer any change at first; that in the large vessels the gelatinous layers mostly form at the points whence branches are given off; that they are more frequent and numerous in the smaller arteries at a distance from the heart, and where the circulating current has less power; that they originate on the free surface of arteries only, and never (as some anatomists assert) between the membranes. Bizot regards them as a morbid secretion of the inner membrane engendered by inflammation.[1]

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[1] For the means of furnishing an accurate anatomical description, we are mainly indebted to the researches of Bizot, who examined the whole arterial system with the utmost diligence in 152 subjects. (Mémoires de la Société Méd. d'Observation, vol. i, p. 262.) The description which follows agrees, generally speaking, with that of Bizot; my own investigations, so far as they go, tending, with few exceptions, to corroborate the far more detailed account given by that pathologist.

Conjointly with these soft deposits are found, occasionally in the same subject, others more solid, and at the same time more firmly adherent to the internal membrane; others of still greater consistency, being whitish and more opaque; others again, closely resembling boiled white of egg; and finally, membranaceous patches almost cartilaginous. Thus have we the means of tracing, though not *always* in the same individual, the progressive transition from the gelatinous layer to the semi-cartilaginous patch, proving almost beyond a doubt the development of the latter from the former. This is moreover corroborated by the experience of Bizot, who met with the softer deposits more frequently in young subjects, and the harder in old ones. It is seldom possible to discover any trace of internal membrane beneath these patches; for although, with care, we may succeed in separating this membrane along with its gelatinous investment, it usually tears off at the confines of the cartilaginous patches, proving the latter to be in immediate contact with the middle tunic of the artery. In some preparations, however, I have distinctly observed the internal membrane remaining unchanged beneath the patches. These semi-cartilaginous plates occasionally become partially loosened from their base, and project into the caliber of the artery, thereby promoting the deposition of fibrinous coagula upon their prominent margin. I have observed this twice in the abdominal aorta.

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[1] In evidence he adduces three cases in which (death being preceded by febrile phenomena and by general œdema) the gelatinous layer was found diffused throughout all the arteries. In the first case, the cortical substance of the kidneys was very emphysematous, tearing, on the fibrous membrane being removed, and being of a greenish hue; the tubular substance firm and violet-coloured. In the third, no other organ presented any change except the kidneys. These did not exceed one half their ordinary size, their cortical substance being pale, uneven at the surface, and remaining adherent to the capsule on attempting to remove the latter. We are here somewhat reminded of Bright's granular kidney, to the diseased condition of the blood in which we might be tempted to refer the gelatinous deposition found in the blood-vessels.

These semi-cartilaginous patches undergo hardly any change in the sequel, appearing only to acquire greater thickness. They *never* ossify, although Andral and others have maintained the contrary. Where several patches are grouped together, the whole surface of the artery, for a considerable extent, becomes coated with a thin, pellucid false membrane, giving it the semblance as if the inner membrane consisted of several layers, and was prolonged over the semi-cartilaginous patches. The arterial walls then acquire an unusual thickness, become stiffer, and less elastic, and their free surface, without losing its polish, assumes a delicately puckered character.

The deposition of *atheroma* is a very different process. This substance originates between the internal and middle membranes, or else betwixt the fibres of the latter. In its ulterior changes it destroys first the middle and then the internal membrane, leading eventually to the so-called ossification of the vessels.

The incipient signs of this morbid process are cognizable in small, more or less sharply defined, roundish patches of the same yellowish white colour as the artery, only more intense. In youngish individuals these spots are most commonly found in the thoracic aorta, where, near the origin of the intercostal arteries they are arrayed in two interrupted lines descending along both sides. At first they are perfectly flat, but after a while slightly raised. If, under these circumstances, the internal membrane is removed, a whitish substance, somewhat like boiled white of egg, partly comes away with it, partly clings to the middle membrane, which latter appears, at such spots, of a light yellow colour, of rather friable texture, and of a structure much less distinctly fibrous than natural.

The degeneration seldom remains stationary at the above grade, but spreads superficially, and frequently over a considerable extent of the arteries. Further changes peculiar to middle and to old age afterwards supervene. The mass generated between the internal and middle coats softens and increases in quantity, occasioning gibbous prominences on the free surface of the arteries. The morbid product is then found in every degree of consistency, from that of boiled white of egg to that of pus,—its appearance being, however, generally that of thick pease porridge. Bizot has often detected in it shining particles, like gold dust, and Cruveilhier accumulations quite similar to the cholesterin scales of certain gall-stones. These masses have been examined by Gluge under the microscope, and recognized as congeries of fat-globules.[1]

In proportion as the emollition proceeds, the middle membrane softens and liquefies, until a mere remnant of its outermost fibres remains sound, prone likewise to eventual destruction. The internal membrane resists tolerably long, but ultimately cracks, and allows a gradual escape of the fluid portion of the subjacent accumulation. In many instances, it strips off from the whole of the diseased patch, giving rise to a more or less diffuse ulcer, the base of which, formed by the external, and by what remains of the middle tunic, is washed by the sanguineous current. Ulcers of this description are frequently met with in numbers in the aorta (especially the abdominal aorta), but are very rare in the smaller arteries. In the pathological museum of the Leipsic hospital, there is an aorta exhibiting a continuous series of ulcers through its entire length. They are of very irregular shape, with flat acuminated ragged edges, and a very uneven base. Ulcers of this nature may sometimes lead to sudden death, through perforation of the arterial membranes.[2] No trace either of inflammation or of vascular development is discoverable in the middle and internal membranes in the neighbourhood of the ulcer; their edges, however, and also in some measure their base, display a rather intense gray or black colour, and their circumference is beset with little fibrinous coagula, which sometimes accumulate in considerable stratiform masses. The cellular layer is implicated in all these changes, getting thicker in proportion as the atheromatous degeneration advances, and at the same time becoming the seat of a tolerably dense web of minute vessels.

The substance deposited between the internal and middle membranes may, however, instead of softening, undergo the opposite process, namely, that termed *ossification.* It is, indeed, not rare for one part of a disorganized patch to ossify, and for another to become destroyed by softening, whilst the first grade of atheromatous disease is extending its ravages to contiguous portions of the artery. Ossifications of this kind mostly assume the form of thin and very brittle layers of a pale yellowish colour. The internal membrane passes over them unchanged; sometimes however it disappears, so that the stream of blood comes into immediate contact with the bony plate. It enlarges at the expense of the middle membrane, whose fibres become compressed, more and more attenuated, dried and atrophic; or else soften and waste away, owing to a semi-fluid atheromatous mass deposited beneath the ossification. Although the bony scales adhere pretty firmly to the arterial membranes at their margins, yet they sometimes crack and tear at the centre, thus favouring the ulcerous disposition of the parts, and projecting in their partially loosened state into the cavity of the vessel. Frequently they enlarge in all dimensions, and form scabrous projections upon the inner surface of the larger trunks.

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[1] I have myself repeatedly examined them with the microscope, and found them, on several occasions, to consist of fat-globules merely; generally, however, they were made up of an amorphous granular mass, mingled partly with fat-drops, partly with numerous cholesterin rhomboids.

[2] Arch. gén., vol. iii, p. 569, and Journ. de la Soc. de Méd. de Montpellier, Mai 1840.

Valentin,[1] who has submitted to microscopic examination the morbid, bony, and earthy concretions of the human subject, has shown that the so-called ossifications of vessels differ essentially from true bony substance. He terms them organized calcareous deposits; inasmuch as the earthy particles are deposited as round, annular, or irregular compact bodies (with prolongations radiating in all directions) within a pellucid, more or less lamellated, and finely granular organic texture.

In rare instances, the atheromatous mass is reduced by absorption, and the ulcers heal, without any occurrence of ossification at such spots. The internal surface of the artery exhibits blackish pits, with scar-like puckering and attenuation of all the membranes.

The several forms and degrees of atheromatous disease are found not only often intermingled in one and the same locality, but also not uncommonly associated with semi-cartilaginous patches, so that a superficial observer might regard *these* as the transition stage to ossification. We have, however, seen that the two products are essentially dissimilar, as respects both their seat and mode of development.

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[1] Repert. für Anat. und Phys., vol. i, fasc. iii and iv, p. 317.

Atheromatous deposits occur in the smaller vessels as well as in the aorta, with this difference, that in the former, the transition into complete softening and ulceration is comparatively rare. On the other hand, the morbid secretion may be so abundant, at certain points, as to stop up the caliber of the vessel.[1] In the aorta, coarctation or obliteration is, for the reasons above assigned, to be looked upon as a rare exception ; indeed, the simultaneous development of cartilaginous patches and of atheromatous changes, tends rather to produce dilatation. At all events, a diminution or loss of elasticity in the arterial walls must result both from the total, or almost total, destruction of the middle membrane, and from the production of semi-cartilaginous masses, which are almost entirely devoid of elasticity. The aorta is thus converted into an inert tube, hardly if at all sensible to the impulse of the blood. Hence the origin of many a dilatation of the aorta, without aneurismal sac, in which cases, there are always numerous cartilaginous patches, besides extensive atheromatous degeneration, —the cellular tunic alone remaining unchanged. A delicate false membrane invests, for the most part, the semi-cartilaginous patches, but differs in consistency, colour, and transparency from the innermost coat, and is not continuous with it.

Besides the above described variety of ossification, another is met with in the smaller arteries, especially of the lower extremities, unconnected with atheromatous deposition, as accurately defined by Andral and Lobstein. Here the internal membrane is rarely involved, the disease confining itself to the fibres of the middle coat. This coat,—which in the sound condition presents a smooth dense layer of fibres intimately interwoven,—loses its equable aspect, while some of its fibres waste, shrivel, assume a finely granular surface, and instead of being elastic, are friable and easily torn. Hence the arteries appear externally, but more especially internally, wrinkled, puckered, and distinctly dilated. When cut lengthwise they are not easily spread out, and the incised edges appear as it were fringed or jagged ; obviously the result of the modified condition of the fibres of the middle coat, some of which still retain, whilst others have parted with their elastic character. If the majority of the fibres become atrophic and brittle, they give way in some places, causing the artery to dilate unequably, and to acquire an attenuated, and here and there constricted appearance. This change is most frequently observed in the iliac, and especially in the crural arteries. In many cases, there is deposited in these atrophic fibres phosphate and carbonate of lime, as a congeries of granules—converting them into osseous bands which ultimately form a complete ring round the arterial caliber. This kind of ossification may affect the arterial system to a great extent, spreading along the course of whole arteries, together with their branches, and imparting to their inner surface a rough granular appearance.

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[1] As figured in Cloquet's Pathologie Chirurg., pl. ii.

Finally, Bizot[1] has observed, in the tibialis antica, postica, and the peronæa, a third very rare description of ossification, consisting in minute calcareous granules, which form in linear direction upon the lining membrane, at first in elliptic groups, but by and by in irregular scales. They are originally deposited around transverse fissures of the internal coat.

It has already been stated that the morbid changes under consideration vary in frequency in the different arteries, and that in some they are not met with at all. Thus the several types of the disease are found chiefly in the posterior surface of the aorta, and the ulcers almost exclusively in the abdominal aorta. The secondary forms (atheromatous softening and ossification) as affecting the different arteries, have been classed by Bizot in the following order of frequency: tibialis postica, carotis cerebralis, tibialis antica, peronæa, subclavia, coronaria cordis, poplitea, cruralis, iliaca communis, radialis, anonyma, brachialis, axillaris, ulnaris, carotis facialis, carotis communis. In reference to ossification, Lobstein[2] gives the following arrangement: arcus aortæ, extremitas iliaca aortæ, aorta thoracica, lienalis, aorta abdominalis, cruralis with its branches, spermatica, hypogastrica and its branches, coronaria cordis, branches of the subclavia, bifurcation of the carotis communis, convolutions of the carotis cerebralis, branches of the carotis externa, arteries of the thoracic and abdominal parietes, brachialis and its branches, twigs of the umbilicalis, arteries of the substance of the brain, pulmonary artery.

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[1] Loc. cit. p. 346.

[2] Traité d'Anat. Path., vol. ii, p. 558.

A very remarkable observation has been made by Bizot, namely, of the *symmetrical occurrence* of arterial disease as an absolute law. Thus the same morbid changes are wont to develop themselves in the corresponding arteries of both halves of the body at the same point, and in equal degree and extent.

The *sex* does not appear to exert any material influence upon these affections, except that in females they are developed at a later period of life, and in a minor degree. With them ossification is more common in the abdominal aorta at its bifurcation, and at the root of the inferior mesenteric artery, as also in the arteries of the upper extremities; whilst in males the coronaria cordis and the vessels of the lower extremities are far more liable to the disease.

*Age*, on the contrary, is in this point of view of most decided import. Each morbid phenomenon progressively increases in frequency and extent, with the age of the subject. Thus we meet in young persons with the primary stages of development of the disease alone, whilst in old ones these are found associated with the more matured forms. Instances are on record[1] of ossification of arteries, more or less extensive occurring in childen of 15 months, of 3 and 8 years, at the age of 18 and of 24; still they are rare, and rather to be regarded as exceptions. However commonly ossification is met with in old age, it is by no means to be considered as its necessary accompaniment, there being numerous examples to the contrary.[2]

In the present state of our knowledge it is hardly possible to determine on what morbid process all these organic changes depend. Andral and Lobstein connect them with a peculiar taint in the fluids, closely resembling the arthritic. At all events they are to be considered not as a local disease, but as referrible to some constitutional cause.

On the other hand, pathological anatomy has furnished very striking illustrations of the effects produced by atheromatous deposit in arteries, upon the organs they supply. It has been stated, that in the smaller arteries the atheromatous mass is deposited in such wise as to occasion more or less coarctation, if not partial closure of the canal. This is most frequently observed in the arteries of the leg, of the brain (especially in the arteria fossæ Sylvii), and of the heart, and it is accompanied at certain points by ossification of the arterial walls. These circumstances combined serve to diminish, and, if the stoppage happen in several branches at once, entirely to cut off the supply of blood,—the natural consequence of which is atrophy, and eventual mortification of the parts implicated. Thus where the coronary arteries are affected in the manner described, atrophy of the substance of the heart is a usual phenomenon. Many instances of gangrene of the lower extremities, and a certain form of cerebral softening are probably due to a similar cause.

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[1] Andral (Précis d'Anat. Path. Brux. 1837, vol. ii, p. 64.)

[2] Ibid. Lobstein, vol. ii, p. 560. Albers Erlänterungen zu seinem Atlas der Path. Anat., Part 3, p. 161; Bouillaud Dict. de Méd. et Chir. Prat. (Art. Cœur.)

### SECT. III.—DILATATION OF ARTERIES. ARTERIECTASIS. ANEURISM.

Aneurism was formerly regarded as of almost exclusively mechanical development, and it is still customary to arrange under this head phenomena of a very dissimilar kind. In proof of this we submit a general view of its classification, together with the synonymous nomenclature.[1]

1. *Aneurysma verum,* all the three arterial membranes being equally dilated.
    1. An. verum totale, comprehending the whole arterial tube.
        - *a.* fusiforme } diffusum.
        - *b.* cylindroideum } diffusum.
        - *a.* Aneurysma anastomoticum (Bell); by others called aneurysma per anastomosin. The branches of several arteries all dilated, forming amongst each

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[1] This nomenclature has given rise to much confusion, certain forms having been variously denominated, and again the same name given to several distinct forms: thus, the terms, aneurysma per anastomosin, aneur. spurium circumscriptum, &c., have been used to denote sometimes the same, sometimes dissimilar affections. Cruveilhier, aware of the imperfections of the older classification, and sensible, from the frequent anatomical inspection of aneurisms, of the difficulty of ascertaining the exact condition of the arterial membranes, proposes a classification based entirely upon outward form, viz.: *A.* Aneurismes sous l'aspect d'ampoules: 1. An. Périphériques; 2. An. Sémipériphériques; 3. An. à Bosselures. *B.* An. sous l'aspect de Poches à Collets, upon which the varieties 1, 2, 3 might be aptly, and, so to speak, naturally, ingrafted. (Livr. 28.)

other a multiplicity of communications, and collectively one common tumour.
    - β. Telangiectasis (Gräfe), Tumor erectilis, (Dupuytren.)
  2. An. verum partiale.
    - a. an. secciforme (circumscriptum.)
    - b. an. cirsoideum. Varix arterialis, (Dupuytren.)

II. *Aneurysma mixtum.*

1. An. mixtum externum,—internal membrane lacerated,—middle and external distended.
2. An. mixtum internum. An. herniosum, (Dupuytren,)—middle and external membranes torn, internal protruded.

III. *Aneurysma spurium.*

1. An. spurium sacciforme, circumscriptum. Internal and middle membranes destroyed,—external membrane composing the aneurismal sac.
2. An. spurium diffusum, primitivum et consecutivum.—Circumscribed extravasation, from the lesion of an artery.
3. An. spurium varicosum, varix aneurismaticus. Aneurisma per transfusionem, (Dupuytren); an. per anastomosin. An artery and a vein communicating in consequence of a wound common to both.

The above classification obviously rests, for the most part, upon outward form and other accidental characters, rather than upon a fixed pathological basis. In the following description, aneurismal and cirsoid aneurism, with telangiectasis, will specially be considered according to their distinctive signification; while diffuse spurious and varicose aneurisms, being of traumatic origin, will be noticed only in as far as they serve to throw light upon true aneurism, the production or consequence of internal disease.

All varieties of aneurism, except those alluded to, correspond in the more or less diseased condition of their arterial membranes; unless, indeed, those cases are to be reckoned in which the aorta is at its origin dilated to a circumference of between three and four inches,[1] (the membranes being nearly if not quite sound,) and in which, somewhat higher up, the canal bulges out towards the left,—phenomena[1] commonly depending upon old age or disease of the heart. The progressive formation of the true fusiform, of the cylindroid and sacciform, of the mixed (if admitted to exist) and of the spurious sacciform aneurism, may be traced to the morbid changes of the arterial membranes described in the last chapter.[2] In cases of aneurism, however, more extensive degeneration of these kinds occurs about the dilated part than is otherwise met with, except now and then in very aged persons. I have ascertained this fact by the examination of fresh aneurisms, of others preserved in alcohol, and, lastly, of a numerous collection of dried preparations, and found it confirmed by detailed reports given by other observers. In every instance that has come under my notice, the arterial membranes were covered with semi-cartilaginous patches and false membranes, rendered uneven, softened or partially exulcerated by atheromatous deposits, and extensively ossified.

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[1] Three inches $5\frac{1}{2}$ par. lines (in a male of 70 years) is the greatest width out of 120 measurements made by myself.

If we bear in mind the multifarious changes which the internal and middle membranes were, in the preceding chapter, seen to undergo in these processes, we shall have less difficulty in accounting for the discrepant opinions of different authors, as to the *possible* occurrence of certain forms of aneurism.

We have seen that atheromatous degeneration and its various sequelæ, completely, or almost completely, destroy the internal or middle coats of arteries within a greater or less extent; and also that the semi-cartilaginous patches usually coexistent, taking the place of this internal membrane, render the canal stiff, fragile, and often preternaturally thick. Should all this disorganization occur around the artery equably at one spot, it will become devoid of elasticity, and in short, have nothing healthy about it except its filamentous sheath. The column of blood acting with equal force on every side, will now gradually determine a slight dilatation at the points farthest advanced in the process of softening. If the whole circumference of the canal is involved, the softened parts will give way here and there, and be again partially consolidated, till the aggregate of these several dilatations constitutes one uniform aneurism, which becomes *cylindrical,* provided the artery immediately above and below the dilated portion resumes its healthy character, but *fusiform,* where the transition from the diseased to the healthy state is gradual. Such will no doubt be a *true* aneurism, inasmuch as the external membrane is not exposed, and does not form the only covering to the tumour, remnants of the middle and internal membranes being still everywhere present; it would indeed appear as if these membranes, whether thickened, or attenuated, hardened into cartilage, ossified or softened, still continued to invest the whole sac; for on a superficial inspection, the atheromatous mass may in several of its stages of degeneration be mistaken for the middle membrane of the artery, since the internal membrane is partially replaced by the semicartilaginous patches, and the whole surface generally invested with a thin, smooth, semi-transparent false membrane. Such are the aneurisms in which all the membranes have been erroneously assumed to have undergone uniform distension; but where, in point of fact, the two inner ones (as just pointed out, and as Hodgson has shown in opposition to Scarpa) may still *appear* to exist, but are in reality for the most part destroyed.

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[1] The dilatation of anastomotic branches, after closure of large arteries, or of the internal spermaticæ in pregnancy, have no connexion with aneurism.

[2] A. Scarpa (Sull' Aneurysma, &c., Pavia, 1804); J. Hodgson (Diseases of Arteries and Veins, London, 1815); Laënnec (Traité de l'Ausc., t. iii); Cruveilhier (livr. iii, xvii, xxviii); Bizot (loc. cit.); Cerutti (Description of the Pathological Preparations of the Anatomical Theatre at Leipsic, Nos. 735, 766.)

Where those degenerations are not equably distributed around the whole circuit of the canal, but where the softening is almost confined to one side, it is easy to conceive that such gradual distension may lead to the establishment of an extensive pouch, formerly supposed to contain all the arterial membranes, the false membrane before alluded to having been mistaken for the internal coat. These relations, particularly as regards the delicate false membrane, have been most clearly elucidated by Bizot. Hodgson, by whom this form is duly recognized, states that when the sac becomes at all extensive, the individual membranes are by no means traceable in an entire state throughout, but only partially, and to a short distance from the entrance. For the reasons before alleged, Lobstein considers it extremely difficult to demonstrate the individual membranes of aneurismal sacs.

The forms just described are, upon the whole, rare. Scarpa appears not to have met with them at all. I have seen comparatively few decided examples of spindle-shaped aneurism of the aorta and its principal branches.

These *true* aneurisms can of course rarely acquire a considerable size, without bursting either completely or partially, (in which *last* case, they give rise to sacciform spurious aneurism,) or else causing the fibres of the elastic tunic materially to give way. Nor do the fibrinous coagula about to be described become deposited within their cavities, although exceptions to this rule are said to have been observed (vide Burns and Hodgson.)

*Mixed* aneurism has been a fruitful source of controversy. It was first pointed out by Haller, and since his time, there has never been any want of cases either to confirm or invalidate its existence. William Hunter was its most powerful supporter; yet its possibility has been called in question, chiefly on the authority of Scarpa's researches; so that when Dubois and Dupuytren sought to demonstrate the reality of internal mixed aneurism (*aneurisma herniosum*), their case was received merely as an exception. Breschet [1] conceives that he has satisfactorily proved its occurrence in smaller arteries; the subject however still needs farther observation and research: for mixed aneurism implies, as a fundamental condition, great capability of extension, *without rupture,* of the middle and internal membranes, a property directly the opposite of their real attributes.

John Hunter and Everard Home [2] experimented upon animals, with a view to ascertain whether the force of the circulating current sufficed, after destruction of the external membrane, to cause the middle and internal, or after destruction of the external and middle, to cause the internal membrane to protrude aneurismally; they found, however, that the removal of portions of those membranes, instead of inducing aneurism, was followed by the effusion of plastic lymph, and healing of the part, without any change in the caliber of the artery. Against these experiments it might be urged, that being instituted upon healthy vessels, the results could not hold good with reference to morbid conditions; it must not, however, be forgotten, that in disease, the middle and internal membrane become almost wholly deprived of their elasticity, and are then, as has been already hinted, more prone to tear than stretch. An instance occurred at the Leipsic hospital of syphilitic caries of the cervical vertebræ, producing softening and perforation of the right vertebral artery. The opening in the latter was small and funnel-shaped, so that the injury of the cellular membrane was greater than that of the fibrous one. There was no protrusion whatever of the internal membrane; the edges of the minute perforation in it were sharply defined and not everted; yet the blood had become extravasated beneath the periosteum and the ligaments of the common vertebræ, constituting a so-called *diffuse spurious* aneurism. Similar instances have been recorded by others.

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[1] Mém. Chir. sur diff. espèces d'Aneur. 1834.

[2] Transactions of a Society for Improvement of Med. Knowledge, vol. i, p. 144.

In deducing inferences from anatomical preparations, we should by no means lose sight of Bizot's remark, that what frequently appears to be the internal membrane of the artery, spreading out so as to line the aneurismal sac, is in reality only a somewhat analogous false membrane, investing not only the arterial surface, but likewise the distended cellular tunic, and occasionally certain fibrinous coagula deposited here and there within the sac.[1] It is, moreover, known that a current of blood getting beyond its natural bounds, strives to establish a new barrier; and that in the first place by a new formation nearly resembling the common membrane of blood-vessels. This event attends upon the developing of new vessels, the circumscribing of diffuse spurious aneurism, &c. I have never met with an aneurism in which this adventitious membrane was not present, and have often seen it lining diseased arterial tunics. Carswell has so figured it (Fasc. Analogous Tissues. Pl. ii, Fig. 8) in an instance where it had become the seat of calcareous deposition, which, as this membrane is unorganized, must have come directly from the blood. Where no mention is made of this membrane, some doubt may be entertained as to the real existence of mixed aneurism.

The most frequent and generally recognized form is that of

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[1] A case deserving of notice is given by Wardrop in his work on Aneurism, London, 1828. In an aneurism of the anonyma the entrance into the right carotis was found closed by a thin membrane, apparently a continuation of the spurious one lining the sac.

*circumscribed spurious* aneurism. Here laceration or destruction of the internal and middle membranes is followed by sac-like distension of the filamentous sheath. It has been artificially produced by Nicholls and Hodgson,—the latter, first dividing both the inner membranes by the application of a ligature, then removing it again, and causing the external coat to protrude in a sac like manner by the inflation of air, or the injection of fluids. Rupture never takes place when the inner membranes are in a healthy state, as is proved by general experience, as well as by the direct experiments of Jones.[1] That accident is always preceded by the degeneration before alluded to ; the portion of the arterial tube which gives off the aneurismal sac, being almost always equally or unequally dilated in its whole circumference. Then either increasing fragility of the middle layers leads to rupture, (mostly transverse, rarely longitudinal,) or else there occurs in the progress of softening and ulceration, so much loss of substance as to lay bare the external coat, and render it liable to saccular distension from the force of the sanguineous stream. The former process has been carefully studied by Scarpa, the latter by Bizot.

The sac while gradually enlarging pushes forward the surrounding cellular tissue, and the neighbouring parts, and thickens at their expense, and the thickening is promoted by an increased, almost inflammatory vascular action in the sac itself; its parietes assuming a sero-lardaceous or fibro-cartilaginous character, in which the cohering membranous textures partake. Under these circumstances, there sometimes occur ossifications of the sac, but quite distinct from the calcareous depositions upon its inner surface. The latter, Bizot has shown to originate in the atheromatous masses, frequently generated within aneurismal sacs, and liable just as within the entire artery, to soften and to ossify. If, on the other hand, the aneurismal tumour presses against very solid parts, such as cartilage, and especially bone, its parietes almost invariably waste by slow degrees, and finally disappear. The periosteum interposed between the sac and bone, being simultaneously absorbed, the fibrinous layers about to be described within the aneurismal cavity, impinge upon the true substance of the bone. At the edges of the parts in contact, the aneurismal parietes afterwards mostly very firmly coalesce with the thickened periosteum, and with the neighbouring soft parts. In every instance, however, fibrinous coagula form in concentric layers within the sac. These various layers are the more solid, and evidently the older, the more external they are, so that in examining each in turn from without inwards, we are generally enabled by their consistency and colour, to trace the gradual transition from the firmest fibrinous layer,—having at times almost an organized aspect,—to recently coagulated blood. This last, however, does not always form the nucleus, inasmuch as the older layers when imperfectly connected together, leave interspaces, into which fresh blood is poured by the artery, occasioning an alternation of old and new layers.[1] These fibrinous formations so highly important as regards the pathology and treatment of aneurism, are, in all probability, deposited directly from the more or less stagnant blood within the sac; similar fibrinous layers surround the extravasation in diffuse spurious aneurism. Kreysig may possibly be correct in supposing a concurrent inflammatory condition of the membranes to cooperate in their development; but Schönlein's idea of their being the essential constituent of aneurism, and of a fungoid carcinomatous nature, is wholly untenable.

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[1] On Hemorrhage, p. 125.

The shape of the sac depends, in most instances, upon the nature of the surrounding parts; in the cavities of the body it is usually spherical; its greatest convexity, as Everard Home remarked, inclining towards the course of the artery. The sac, at its origin, is contracted, and attached by a pedicle or neck, narrow where the aneurism proceeds from laceration,—but wide, often more so indeed than the sac itself, where it arises from ulcerous destruction. In the former case, the edges of the orifice are uneven, rough; the middle membrane prominent like a torn fringe, often exhibiting calcareous deposits, whose sharp points encircle the gap created by the rupture.[2] In the latter case, the opening is rounded off, smooth, and more regularly spherical or oval; the inner membranes become gradually extinct, and the whole surface is invested with a smooth, thin false membrane, making it extremely difficult to ascertain the anatomical condition of the individual arterial tunics, more especially where the atheromatous deposits and the adventitious membranes are continued into the sac itself.

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[1] This is well illustrated by a figure in Cloquet's Pathologie Chirurgicale (Paris, 1831), pl. 1, copied into R. Froriep's Chir. Kpfrtfln. (Surgical Plates) pl. cclxxxix.

[2] Cruveilhier (liv. xvii) met with an aneurism of the aorta with a double orifice.

Aneurisms may, under propitious circumstances, attain an extraordinary size, getting more and more dilated at the expense of neighbouring parts. Within the skull alone, where the cellular texture far less favours their dilating beyond a certain degree than elsewhere, they burst very soon. Within the thoracic and abdominal cavities, however, and in the extremities, they go on enlarging, until by pressing against the outer integuments, and so causing sloughing, they at length burst, and death ensues. In the progress of dilatation, aneurisms, and especially internal ones, give rise to a variety of disturbance in many organs, and to a multiplicity of symptoms. They coalesce with serous membranes, or with parts having a serous investment; some organs they compress, displace and emaciate; in others they occasion inflammation, suppuration and perforation; by flattening the nerves they cause the parts they supply to become atrophied, or to pass into a state resembling chronic inflammation; and by compressing the veins[1] and arteries, effect their complete obliteration. The branches springing from the dilated artery become, in like manner, obliterated, either from their mouths being obstructed by the fibrinous deposits of the aneurismal sac, or from being plugged up with semicartilaginous patches and atheromatous matter, or from their orifices, or perhaps a portion of their caliber being rendered impervious by the simultaneous extension and pressure induced by the aneurism. The development of a collateral circulation mostly compensates for such obliteration. Otherwise atrophy or gangrenous destruction of the parts implicated is the consequence. The aneurismal sac coalesces with the periosteum, occasioning, by continued pressure and pulsation, the absorption of the bone, so that the most extensive havoc of the ribs, sternum, shoulder-blades, vertebræ, &c. being here independent of caries, is bounded by smooth surfaces. Greater resistance is offered by the cartilages, which often outlast all the surrounding bony substance.[1]

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[1] In a case related by Weisbrod (Obs. Path. præcip. ven. cav. sup. tangent. Monachii, 1831), an aneurism at the arch of the aorta had burst into the superior vena cava, which firmly coalesced with the sac. Beck (Medical Gazette, Dec. 1841, p. 445) observed a case of aneurism of the ascending aorta opening into the right,— Marsh (Dubl. Journ. July, 1841), another opening into the left ventricle of the heart. Several instances have been recorded of aortal aneurisms opening into the pulmonary artery. (Reid, Edinb. Journ. Jan. 1840; Wells, Transact of a Soc. for the Improvement of Med. Knowl. vol. iii, p. 81, 1808.) I have myself seen an example of several small aneurismal dilatations in the right coronary artery, one of which communicated by means of a minute aperture with the pulmonary artery.

The observation of Hodgson, that aneurisms always determine gangrene in the external skin or mucous membranes with which they happen to come into contact, is corroborated by general experience. Thus, fatal hemorrhage ensues from gangrene (the destruction proceeding from without inwards) when the sac reaches the surface of the external skin, or of a mucous membrane; again, from immoderate distension of a softer portion of the sac (the destruction proceeding from within outwards) when it coalesces with a serous membrane. In the latter case the blood may discharge itself either into a serous cavity or into the perenchyma of an organ coated with a serous membrane—the pulmonary tissue, for instance. Here there is simple laceration, or rather perforation, without any trace of inflammation of the serous membrane, and the question might arise, whether the perforation be purely mechanical,—the consequence of excessive distension,—or rather dynamical, the result of a softening process in the proper walls of the sac itself. The examination of three recent preparations disposes me to adopt the latter view. In all the three the fatal perforation occurred at points the least liable to be put upon the stretch; in two, at the lower end of the sac, in a direction contrary to the course of the blood. The opening was not elongated, like that resulting from a tear, but roundish, like that of a pulmonary abscess bursting into the cavity of the pleura. Adjoining the perforation there was decided emollition extending inwardly, and chiefly implicating the adjunct fibrinous layers, which, being soaked with blood, were of a pulpy consistency, resembling the softened plug in inflamed veins.

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[1] The anatomical museum at Leipsic contains ample evidence in point. One of the preparations was used by Rosenmüller to demonstrate that, besides the mere absorption of bone, aneurismal pressure may even induce caries. (V. Cerutti's description, &c.)

Aneurism, when left to itself, in most, though not in all instances, proves fatal in the manner above described; occasionally, however, it yields to the curative powers of nature. Hodgson has the merit of having clearly established by facts the conditions under which recovery is likely to occur. According to him, spontaneous cure may happen in three different ways: first, by the gangrene so gaining ground during the advance of the aneurism towards the external skin, as eventually to destroy the sac itself, the artery meanwhile becoming closed by a solid plug. The morbid parts then slough away, leaving a suppurating sore to heal by granulation, whilst the artery is obliterated, up to the origin of the next superior branch. Examples of the kind have been met with in arteries of the extremities. Secondly, by the aneurism being so situate as to compress the artery above, and thus produce obliteration. Thirdly, by fibrinous layers being from some cause or other deposited in such regular order and to such an amount, as to fill, ere long, the sac, and preclude all further entrance of blood. The artery itself may then either fill with fibrinous layers and subsequently become impervious, or, as was sometimes found by Hodgson, and more recently by others, its caliber remains free, and the blood passes unimpeded over the sac, almost thoroughly closed. I have myself witnessed this in a tolerably large aneurism of the ascending aorta, in which, during the patient's life, no evidence of this, in ordinary cases, readily detected disease, was obtained, even by the aid of repeated auscultation. In the second and third case, the tumour corrugates, and is transformed into a closed sac, which, by the gradual absorption of its contents, becomes progressively smaller and harder. The circulation of the parts involved is at length restored through the development of collateral branches.

Bizot has made the interesting observation, that the frequency of aneurism in different arteries is, in some measure, dependent upon certain variations in form, first noticed by himself in the canals of various arteries. Thus, of the innominata, the common carotid, the brachial, and iliac arteries, he found the normal shape to be that of two cones with the apices conjoined, ⟩⟨; of the radial and crural, that of the cylinder; and of the poplitea, that of two cones united base to base, ⟨⟩. Of 142 examples, of non-traumatic aneurisms observed by various authors, 75 were of the poplitea, 35 of the crural in the inguinal region, 16 of the crural elsewhere, 11 of the axillary and subclavian, 2 of the external iliac, 2 of the brachial, and 1 of the radial artery. But this statistical survey would seem to require remodelling, for I have myself more frequently met with aneurism of the aorta than of other arteries, an observation confirmed by the high authority of Rokitansky.

Aneurisms sometimes, though rarely, occur in the pulmonary artery,[1] just as the latter, notwithstanding Bichat's denial of the fact, has been seen by various pathologists, and once by myself, partially ossified.[2] Out of 120 individuals examined, this artery was found in ten only to contain inconsiderable grades of atheromatous degeneration, consisting in small slightly prominent yellow spots.

Dilatations of *Botalli's duct* have been observed by Rokitansky.[3] In character they by no means resemble the aneurismal formations hitherto described, being of purely mechanical origin. The duct either does not close towards the aorta, and undergoes through the impulse of the circulation a funnel-shaped dilatation, or else it closes incompletely at one of its two extremities, allowing blood to penetrate and widen its caliber in a sac-like manner.

It would appear that sex exerts considerable influence upon the formation of aneurism. According to Hodgson, the proportion was, out of 63 cases, 56 men and 7 women; according to Bizot, out of 189 cases, 171 men and 18 women.

Out of 108 aneurisms, 1 was observed between the 10th and 19th year; 15 between the 10th and 29th; 35 between the 30th and 39th; 31 between the 40th and 49th; 14 between the 50th and 59th; 8 between the 60th and 69th; 2

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[1] See Albers' Illustrations to his Atlas, abth. iii, p. 208; Ephem. Nat. Cur., dec. iii, ann. vi, obs. 207; Otto's Selection of cases, vol. i, p. 328; Foriep's Notizen, No. 251, 1839.

[2] Carswell, fasc. ix, pl. ii, fig. 5.

[3] In his Handbuch, vol. ii, p. 579.

between the 70th and 79th; and 2 between the 80th and 89th.

On comparing the above statistics with those given in the foregoing chapter, as to the degenerations of the arterial membranes, there will appear[1] considerable discrepancies, not altogether due to any error in the numerical method. At all events, the relative proportions alluded to show that, besides those precursory diseases of arteries, together with the accidental causes furnished by violent muscular exertion, there must in the development of aneurism necessarily be other potential influences at work. Such are commonly reputed, though upon insufficient evidence, to be the syphilitic and the arthritic dyscrasies. And the question still remains, why the membranes of particular arteries, not always the most predisposed to them, should at certain points, become subject to degenerations of a nature adequate to produce aneurism,—and why the male sex should be more especially liable. That those diseases of the arteries and the generation of aneurism have a common cause, is attested by the fact of several aneurisms frequently occurring in the same individual.[2] The experiments of Jones,[3] who never found aneurism produced even by the sudden laceration of a *sound* artery, would tend to prevent our referring the affection to mere local circumstances, such as external violence.

The remarks of Rokitansky,[4] respecting the relations which aneurism bears to other diseases, are highly important: "Out of 108 cases, tubercle coexisted in five only, restricted to a small portion of the lung, and either engaged in the process of retrogression, or altogether extinct. There is an affinity between aneurism and cancer. The aneurismal diathesis is never extinguished; frequently most of the arteries are assailed in turn, and when, from some cause or other, one aneurism dwindles away, a new one immediately forms, either in the same artery or in a remote one. Frequently, too, the aspect of the patient and the decay of the organism bear the impress of cancerous cachexia."

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[1] Those degenerations are alike common in both sexes, increase in frequency with advancing age, are more common in other arteries than in the popliteal (which is most prone to aneurism), and affect concurrently the symmetrical arteries.

[2] Interesting cases of the kind are figured in Cruveilhier, livr. xxviii, pl. i–iii.

[3] Vide supra.

[4] Oesterr. Fahrb., vol. xxvi, pp. 2 and 3.

The consideration of aneurism in general, and of spurious sacciform aneurism in particular, should comprehend the so-called *dissecting* aneurism (*aneurisme disséquante*), first described with accuracy by Laennec, and recently made the subject of still minuter research by Rokitansky,[1] under the name of *spontaneous rupture* of the aorta. Besides Laennec's and one described by V. Stosch,[2] he gives eight cases which came under his own notice; to these, we have to add two by R. W. Smith,[3] one by Nivet,[4] and two by Goddard and Pennock.[5] Here, in like manner, rupture of the internal and middle coats takes place, the cellular tunic is however not distended into a sac by the force of the circulating current, but is separated for a shorter or longer space,—sometimes for the full extent of the aorta,—from the other membranes, the blood meanwhile filling the whole chasm.[6] Rokitansky particularly adverts to the diseased state of the filamentous sheath, which, as the medium of nutrition, necessarily exercises a powerful influence over the two others. He found it, in many instances, of a purple hue; its vessels loaded, and its texture infiltrated and thickened. The rupture was in most instances a transverse one,—occasionally encircling the whole of the arterial canal; in two cases, the upper margin of the fissure was, by the force of the circulation, bent downwards into the arterial tube, intruding upon, or obstructing the origin of the great vessels.[7]

Longitudinal fissures appear to be the consequence of a diseased state of the internal and middle membranes; which latter are found flaccid, easily separable, cartilaginous, ossified, atheromatous. Sometimes there are both longitudinal and transverse fissures at right angles with each other. Cracks traversing in a crooked direction a degenerated portion of the internal membrane of an artery, appear to be the result of the retarded or imperfect development of an aneurism. A rupture of this kind, with separation of the external membrane, may be complicated with an aneurismal sac. (Cruveilhier.) Occasionally the continued impulse of the circulation causes eventual laceration of the filamentous sheath, and effusion of blood into the pericardium and into the cavity of the pleura.

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[1] Oesterr. Jahrb. vol. xxv, fasc. i & ii.

[2] Casper's Wochenschrift, 1834, No. 15.

[3] Dublin Journal, July, 1836.

[4] Bullet de la Soc. Anat. vol. ii, 1836, p. 295.

[5] Americ. Journ. Nov. 1838.

[6] These cases are to be carefully discriminated from those in which all three membranes have given way simultaneously, and blood has discharged itself into neighbouring parts, as Ollivier relates of the pulmonary artery. (Arch. gén. Jan. 1838, Mém. sur les Morts subites.)

[7] Vide plate in Rokitansky.

Rokitansky distinguishes two varieties of these spontaneous ruptures. The first originating in an affection analogous to chronic inflammation in the cellular membrane; separation of the latter being the first effect,—rupture of the internal and middle membranes the second. In the other variety the cellular coat may be quite sound,—but the internal and middle membranes are necessarily diseased. Here the morbid process begins with the solution of continuity in the latter membranes, the parting of the cellular coat through the force of the sanguineous stream being secondary.

The accident occurred in the several cases without previous violent exertion or undue excitement of the circulation,—often in bed, on awaking, &c. Great narrowness of the aortal caliber,—coarctation at the origins of the arteries given off, and a thinness of the membranes,—seem to be the principal predisposing causes. In the case of a youth of 24 years of age, considerable dilatation was discovered at the orifice and in the trunk of the aorta.[1] In every instance the heart was enlarged, the left chamber more especially being dilated, either simply, or with hypertrophy; in Laennec's case it was concentrically hypertrophied; in Stoch's, dilated with attenuation. Out of 15 cases (four others recited by Smith are restricted to anatomical details) 8 occurred in males, 7 in females; 11 in persons between the 48th and 76th year; four in younger individuals,—one in a boy 8 years old and afflicted with morbus cæruleus,—another in a youth of 16 who was subject to hemorrhoids.

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[1] R. W. Smith, l. c.

### SECT. IV.—CIRSOID ANEURISM (ARTERIAL VARIX.)

This form is not dependent upon atheromatous disease, but on the contrary, even precludes the development of that process to a great degree. The latter, generally, as we have seen, enlarges the arteries, causing a number of somewhat sinuous dilatations which render serpentine their course, whilst at the same time it thickens their walls, and that even quite apart from the adventitious deposits. In the *varicose dilatation* of arteries, on the contrary, their parietes are for the most part irregularly attenuate; this form accordingly has a great analogy to phlebectasis. Yet the disease is comparatively rare, and its pathological features by no means well defined.

Two cases are described by J. Cloquet,[1] the one affecting the whole arterial system of a man aged eighty, the other the iliac arteries of a man aged sixty. These arteries presented a considerable widening of their caliber, and a very tortuous course, resembling the intestinal convolutions, with isolated sinuous protuberances and spherical elevations. Their parietes were soft and flabby, the middle membrane peculiarly thin and pale, having lost its yellowish hue, and its distinctly fibrous aspect; in some places it resembled a fibro-cartilaginous layer; it still, however, possessed much elasticity.

Three other cases are detailed by Breschet.[2] In dissecting the body of an aged female, the radial and ulnar arteries were accidentally found dilated and tortuous; the arteries of the hand in particular, were tumefied and dilated in a sac-like manner,—forming a multiplicity of winding anastomoses. In the second case, swelling and induration of the cellular texture in the right leg, together with two aneurisms of the popliteal artery had formed, in consequence of an injury sustained fifteen years previously, in a man aged 65. There existed great dilatation, varicose condition and tortuous course of the tibial artery with all its branches, which, at the foot more especially, presented a dense network of knotted and convoluted anastomoses. In this case, atheromatous changes appear to have coexisted ; for it is said : "the arch and ulterior course of the aorta exhibited dilatations and cartilaginous patches ; the right iliac artery was dilated, and its middle membrane, like that of all the arteries of the right lower extremity, yellowish, and thickened at certain points, at others stretched, in such-wise as to admit the external tunic through interstices formed by the yielding of its fibres."—These are the cases adduced by Breschet, as proofs of the existence of internal mixed aneurism, which *as a mere complication of the cirsoid form* may perhaps be admitted A third example relates to the temporal and occipital arteries, to which might be added many similar instances of aneurismal tumours affecting the arteries of the head, as noticed by various authors.

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[1] Pathologie Chirurg. Copied into Froriep's Chir. Kupfert. (surgical plates), 289; and in Carswell, fasc. ix, pl. iv.

[2] Mém. Chir. sur différentes espèces d'anevrysmes. Copied by Froriep, (l. c. pl. 366.)

I have only once had an opportunity of investigating cirsoid aneurism in its full development in the arteries last mentioned. In the iliac arteries, however, I have frequently met with traces of it in the shape of sinuous bulgings and serpentine windings along the course of the vessels. In all these instances the membranes were strikingly attenuated, owing more especially to atrophy of the yellow fibrous, and in some measure of the cellular membrane. In their characteristic features, the fibres of the former were discernible at intervals only,—having in general merely the aspect of a pale, very dense, but not distinctly fibro-cellular texture. This condition of the middle or fibrous coat may have its foundation, either in a vice of original formation,—which is highly probable in the case of the iliac artery,—or in some affection of the arterial coats,—probably inflammation (chronic, Rokitansky inclines to believe) of the external one, destroying or disorganizing, or else paralysing, as in the case of ordinary arteritis, the middle membrane, and eventually obliterating the vasa vasorum. Disease like this having run its course, might doubtless induce varicose dilatation of the artery.

## SECT. V.—DIFFUSE SPURIOUS ANEURISM AND VARICOSE ANEURISM (ANEURISMAL VARIX.)

Diffuse spurious aneurism and varicose aneurism (aneurismal varix) are not the consequence of internal arterial disease, (except in the case of a spontaneous aneurism opening into a venous trunk), but of accidental, and for the most part traumatic causes.

*Diffuse spurious aneurism* is produced by blood, effused through a wound in the arterial parietes, becoming circumscribed through the resistance of neighbouring parts. This is accomplished chiefly by coagulation of the extravasated blood, aided by consolidation of the neighbouring cellular texture, tendinous structures, &c. The blood does not coagulate in a mass, but, in a series of concentric layers, having at their centre a small open cavity which communicates with the wounded artery, exactly as in spontaneous aneurism. By and by these layers, which were at first of a lively red, become paler and paler, and assume at length a dirty faint brownish colour, verging upon gray.

Sometimes the gradual coagulation extends throughout the cavity of the extravasation, and into the wounded artery, so that the whole mischief may be repaired by immediate *obliteration,* encouraged by suitable treatment. The cellular texture thickens and hardens around the coagulated layers, gradually blending with the latter, and the whole ultimately shrinks into a hard fibro-grumous mass.

In most instances, however, the extravasation very speedily produces a more violent degree of inflammation in the surrounding parts, conducive in the latter to *suppuration,* and in the extravasated mass to ichorous decomposition; whereupon, amid repeated hemorrhage, death ensues, unless averted by the timely application of a ligature above the wound. This violent inflammation, however, seldom terminates favorably, but spreads along the artery, producing extensive adhesion and obliteration.

*Spurious varicose aneurism* is the consequence of a communication between an arterial and a venous trunk, occasioned by both being wounded at the same time. It is most common at the bend of the arm. This communication is sometimes direct, namely when both vessels are closely approximated, or when the lips of the two wounds coalesce. It is, however, more frequently indirect, a clot of blood establishing itself betwixt both vessels, and behaving for the most part, as in diffuse spurious aneurism. The immediate effect of this new and unnatural state of things, is invariably direct ingress of arterial blood into the venous trunk; regurgitation of the whole column of blood in the latter; diminished supply of arterial blood beneath the level of the wound; impeded return of blood from the parts nourished by the artery, and saturation of those parts with a mixed arterial and venous fluid.

The vein suffers various and peculiar changes, originating at the wounded part. First of all it widens, immediately opposite the injury, into a variously-sized and mostly thin-walled sac; similar sac-like dilatations form at different points in the ulterior course of the vein and of its branches. Besides this, there ensues an equable cylindrical dilatation of the implicated venous canals, whose parietes become thicker and stiffer, their inner surface wrinkled, chiefly through unequable thickening of the internal membrane.

Below the point of lesion the artery, together with its branches, become narrower and more thinly coated like a vein, probably from their now receiving a diminished afflux of blood. Breschet has, however, in one instance, found the artery and its branches dilated, and he believes that such may be a consequence of the transmission of venous blood into the artery, during diastole. Above the wound, the artery is usually somewhat widened.

In consequence of the preternatural supply of a mixed nutrient fluid, the adjacent parts suffer various changes. They frequently swell, become œdematous, and peculiarly disposed to inflame. Sometimes they waste away, and functional disturbance follows,—muscular power, in particular, being impaired. The superimposed skin has a reduced temperature and a cyanotic hue, and becomes thickened. Its epidermis scales off or accumulates into knotty or warty masses; and sooner or later, in consequence of the frequent recurrence of inflammation, obstinate ulceration sets in.

## SECT. VI.—VASCULAR GROWTHS.

We have hitherto considered a variety of forms of dilatation and enlargement affecting the whole or certain portions of the arterial system, connected rather with attenuation or wasting of one or more of the membranes, than with morbid deposition between them. We now approach an order of diseased conditions, in which the artery, as such, no longer exhibits changes of structure, but becomes the seat of adventitious and vascular growths, which, under propitious circumstances, may assume a carcinomatous character. To this head belong *anastomotic aneurism, telangiectasis,* and *fungus hæmatodes.*

*Anastomotic aneurism,* or aneurism by anastomosis, was first accurately described by John Bell. It proceeds undeniably from the arteries, and consists in a dilatation of the smaller ramifications, whose numerous, but in the ordinary state slightly visible anastomoses, dilate in such-wise as collectively to constitute a single strongly pulsating, and more or less circumscribed tumour.[1] It is situate deep beneath the skin, and its origin is easy to demonstrate in the larger arterial branches. The corresponding veins, little altered in dimensions, are separated from the conglomeration of dilated arterial branches, by a true capillary system. In some instances, the disease gradually involves this intervening capillary web, and eventually the veins themselves, which soon after begin to dilate.

*Telangiectasis,* on the other hand, is developed out of the capillary system, the dilatation being in the majority of cases manifestly congenital. It therefore exists from earliest childhood, often continues long at its primitive stage, and is susceptible of various changes of form. For an intimate acquaintance with this affection we are indebted to Graefe and Walther.[2]

Although many instances of telangiectasis obviously depend upon dilatation of the capillaries alone, and neither veins nor arteries can be distinctly recognized in the tumour, cases occur, nevertheless, in which the arterial or else the venous character preponderates. In the *arterial form* the tumours are of a bright red,—flattened, and studded with granular elevations resembling in form and colour a ripe strawberry; they tend to spread superficially. After death or extirpation, without collapsing, they become blanched. During their excision, a multiplicity of dilated arterial twigs, distinguishable by the blood gushing forth in capillary jets, are observed to occupy the whole circumference, including the sound skin in the vicinity. In the *venous form*, the tumours are bigger than the others; spherical, knobby, prominent; their surfaces smooth; of a blueish-red or cherry colour; they are prone to enlarge, not only superficially, but in all directions. After death or extirpation, they shrink together disproportionately to their size, partially retain their colour, and convey when compressed between the fingers a certain impression of thinness in their membranes. To this variety belong the angiectases developed in adults; and simple capillary dilatations of long standing, are wont, in middle-aged persons, either subject to hemorrhoids or possessing the "venous habit" of body described by Puchelt, to put on the same character. These venous telangiectases accord in every respect with the so-called erectile tumours of Dupuytren.[1] Andral compares their structure to that of the spleen and the placenta; they bear, however, but a faint resemblance to either.

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[1] It is doubtful whether the aneurismal tumour described by Scarpa (pl. x, figs. 1 and 2) as occupying the place of the superior apophysis of the tibia, is to be referred hither. It is probably rather one of the many transition forms.

[2] Von Graefe (Angiektasie, u. s. w. Leipzig, 1808); Von Walther (Journal für Chirurg. and Augenheilkunde, vol. v, and System der Chirurgie, 1833.) Bouchacourt (sur les Tumeurs érectiles, Rev. Méd., Août 1838, p. 223.)

*Their internal structure* consists mainly in the immediate passage of dilated arterial twigs into venous radicles in a state of saccular and cellular enlargement. From these spring numerous little veins, which in their turn become presently dilated in the same manner, and then pursue their ulterior course as either healthy or varicose veins. Thus originates a texture which through dynamical and other causes becomes gorged with blood, and swells like natural erectile tissue, although generally, merely a soft, elastic, compressible tumour. Dupuytren succeeded in injecting the tumour by the arteries, but not by the veins. It was formerly imagined by Pott and others that blood from the dilated vessels became effused into the cellular texture, and that thus the cellular structure was derived; however, upon close examination, the direct transition of the arteries into the pouch-like dilated origins of the veins becomes apparent. Blood cannot here become extravasated except through accidental rupture.

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[1] Of these, Cruveilhier (Essai sur l'Anat. Path., vol. i, p. 131) gives a minute and instructive account, though he erroneously couples hemorrhoidal tumours with them. (Cruveilhier's plates, livr. xxiii and xxx.)

Wherever erectile tissue is developed, the cellular texture or the parenchyma of organs is ere long removed, and the tumour consists exclusively of vessels closely interwoven and intimately communicating with each other. The external skin is elevated, attenuated to the utmost, and at length gives way under hemorrhage. Under particular circumstances, certain of those venous cells described may be attacked with inflammation, mostly of a low grade, inducing coagulation of the contained blood. Stagnation and the like may operate in the same manner, giving rise to the formation of concentric fibrinous layers, which are sometimes converted through calcareous deposition into veinstones.[1]

Telangiectasis occurs most frequently beneath and within the external skin, more particularly where the latter adjoins the mucous membranes. The tumours mostly occur in groups, and sometimes very diffusedly.[2] That they become developed in parenchymatous organs within the body, is attested by Dupuytren, (see Cruveilhier,) Lobstein, and others.[3] Their relative frequency appears to be greatest in the liver.

The development of these capillary dilatations cannot be ascribed to any general morbid predisposition, nor is their presence apt to exert any deleterious influence upon the constitution. Excision effects their permanent removal. They are, in the majority of instances, congenital; their occurrence in the adult is possibly connected with a hemorrhoidal habit. Telangiectasis with venous predominance is that most prone to assume a serious character. It is, however, not to be denied that certain vascular tumours are *essentially* malignant. We allude to fungus hæmatodes.

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[1] A case of telangiectasis of this nature was operated upon by Prof. Carus at the Surgical Dispensary of Leipsic.

[2] A very striking instance is given by Unger. (Beiträge zur Chir. Klinik, vol. i, p. 175.)

[3] Anat. Path. vol. i, p. 324.

The claim of *fungus hæmatodes* to be classed as an independent form of disease has been recently disputed; one party regarding it as a variety of telangiectasis, another as a variety of medullary fungus.

It is pretty generally agreed that some vascular tumours evince a thoroughly malignant character, without any interstitial deposition of the cancer-cells, distinctive of *true* fungus hæmatodes. Anatomically speaking, they seem to me to betray their malignancy not only in the distension of the natural texture of the coats of the vessels, and in the absorption and wasting of the surrounding structures, but likewise in the lardaceous thickening of the cellular texture and other adjacent parts, such as the external skin; in all of which suppurative inflammation readily ensues. Tumours of this kind exhibit a marked tendency to pass into medullary fungus: nay, in the same individual, morbid growths frequently occur, of which some are unquestionably hæmatoid, others medullary-fungoid, whilst others again are met with of an intermediate nature. In one instance, I could discover in several of the tumours nothing but dilatation of the capillaries, whilst others were perfect specimens of medullary fungus, several containing its characteristic cells between the vascular meshes.

Fungus hæmatodes may be said to correspond in anatomical structure with erectile tissue, except that we can no longer discriminate the venous or arterial portions, or the canals and parietes of vessels; the whole indeed more closely resembles a network of dirty red cells and meshes. The tumours are characterized by redundance of growth in all directions, and especially towards the surface of the body, which, so far from being subdued, is only roused into augmented activity by surgical operation. They break of themselves or upon being meddled with, and form discoloured, ichorous, almost constantly bleeding, uneven spongy sores. Themselves the probable offspring of a general predisposition, they tend in their turn to undermine the system at large, becoming the prolific source of similar, or else of medullary tumours in other situations; a circumstance that would certainly seem opposed to the assumption of bleeding fungus being a specific and substantive disease.

# CHAPTER IV.

## HETEROLOGOUS FORMATIONS IN THE CIRCULATING ORGANS.

At the present day the opinion is almost universal that heterologous growths have their source in a constitutional predisposition, upon which, with or without an obvious occasional cause, depends their development in some particular structure or organ, and their dissemination from thence over various parts of the system. The different textures and organs do not, however, evince equal proneness to engender these growths, or to become the original seat of a specific product of the kind. Tubercle, melanosis, medullary fungus, each show a sort of predilection for particular organs, and develop themselves in these according to defined laws, whilst they invade other parts only after the fluids have become generally contaminated. The organs of circulation are in general liable only to become *secondarily* diseased, and even this far less frequently than most other parts.[1] I may mention in passing, that the common forms of adventitious products, tubercle, melanosis, hydatid, cyst, sarcoma, fungus, thus occur within the textures of the heart, and in some few instances proceed to cancerous destruction.[2] Occasionally they have been found overspreading the heart's parietes to such an extent, that even admitting the tumours to have been of tardy growth, it seemed inconceivable how the organ could have continued to contract. During life naught was perceptible, beyond a diminished intensity of the heart's sound and impulse.

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[1] Otto (Path. Anat. vol. i, sect. 183) Bouillaud. The Pathological Museum of Vienna is rich in these curiosities.

[2] Carcassonne, Mém. de la Soc. Roy. de Méd. année 1776. (See Andral, Précis d'Anat. Path.) I have myself met with an ulcerous medullary fungus, occupying the septum which divides the two auricles, and discharging into the left auricle.

A few scattered examples[1] of medullary tumours, found growing with a pedicle from the inner surface of the *heart,* without any coexisting trace of disease elsewhere, show that that organ may be the primary seat of the morbid development. Of isolated productions in the heart, hydatids and encysted tumours are the most frequent.[2] In man the vascular system is almost entirely exempt from entozoa; the *cysticercus cellulosæ* is the only one that has ever been found in the heart. (Rudolphi, Rokitansky.) The *trichinia spiralis,* which infests all the muscles of voluntary motion, is never seen in those of organic life, and consequently not in the substance of the heart.[3]

No well-authenticated example is known of the *arterial membranes* being the seat of heterologous growths. Whatever have been described as resembling such, have been either various forms of atheromatous degeneration, or else tumours intimately incorporated with the filamentous sheath. Encysted tumours would appear, from the cases cited by Otto,[4] to form a solitary and rare exception. This immunity of the arteries has led to the idea of identifying atheroma with tubercle, a groundless assumption, seeing that the two *substances* are essentially dissimilar, and also that arterial degeneration occurs but seldom, and never to any extent in the tubercular habit. Nay, it is the deliberate opinion of Rokitansky, that aneurism and those affections of the heart which are closely allied to, if not dependent upon atheromatous disease; (see the respective sections, cap. iii) exclude tubercle. Still tubercle and atheroma have some pathological features in common; thus, both of them, in the act of liquefying, destroy the contiguous textures, and both of them, in the process of retrogression, become calcareous. It is otherwise with the veins, which, though alike exempt from tubercle and atheroma, are obnoxious in divers ways to fungous growths.

*The veins* may become diseased in three distinct modes:

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[1] See, in particular, Cruveilhier, livr. xxix, pl. ii. fig. 1, 2; iii, 1.

[2] See two examples of acephalocysts in Rokitansky's Handbuch der Path. Anat. vol. ii, p. 465.

[3] Bischoff. Heidelberg. Annal. 1840, vol. vi, fasc. ii, p. 232.

[4] Vol. i, sect. 203, note 3.

first, by the development of fungous and other growths between their membranes,[1] either as the primary manifestation of an incipient morbid tendency, or the secondary one of a confirmed dyscrasy which has already shown itself elsewhere. Secondly, by the proximity of carcinomatous tumours, which coalesce with, compress, and obliterate them, or else produce thickening, or softening, and in certain instances perforation of their membranes. Lastly, fungous growths may penetrate into the channel of veins, where they evince a tendency to advance in the direction of the heart, and to occupy its cavities.

This occurrence of fungoid or lardaceous growths within the veins has excited much attention, and been variously accounted for. Some refer it to the absorption of the heterologous matter by the minutest venous twigs, others to immediate deposition from the blood.[3] The former view is at variance with physical laws. The primitive cells of those growths exceed the blood-globules in size; it is therefore inconceivable that they could traverse, supposing they could enter the closed capillaries, unless indeed the nuclei of the cells be reckoned the germinal principles of disease. Nor will the second view hold good until the proximate elements of fungus can be *proved* to pervade the whole sanguineous mass. With reference to certain cases, indeed, this hypothesis is not devoid of probability; and Rokitansky has promulgated the opinion that the cancerous cachexy when generally diffused through the system, imparts to the blood the peculiar property of determining an immediate deposit of crude carcinomatous substance blended with fibrin in the cavities of the heart, and especially in the veins. That the veins are the vehicles of the cancerous matter to remote organs is evident, from the fact that cancer of organs, tributary to the portal vein, is most frequently reproduced in the liver;—of those tributary to other veins, in the lungs; as likewise from the direct experiments of B. Langenbeck, (see Cancer of the Lungs.) For the majority of cases, however, a much simpler theory will suffice. Those fungoid growths, namely, which are found luxuriating within the canals of veins, always occur in the proximity of, and can be distinctly shown to originate from cancerous tumours affecting other organs.

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[1] Otto, Path. Anat. i, sect. 208.

[2] Particularly Carswell (fasc. "carcinoma"), who has given several admirable figures of this morbid state.

[3] Schmidt's Jahrbücher, vol. xxv, fasc. i, p. 99.

If we take into account the vascular nature of fungous and steatomatous or lardaceous structures, and the thin-coated condition of their blood-vessels, it is not difficult to conceive that the heterologous mass may, by softening and breaking down the venous membranes, make its way into the canal and grow freely in this new space, where nothing occurs to arrest its progress. This view is, moreover, corroborated by the circumstance that fungous growths (according to my own experience at least) never occur in the interior of veins until after the parent tumour has reached the stage of softening. This is especially manifest in the liver. In an instance of cancer of the lung in the stage of softening, I found one of the pulmonary veins, from its origin, completely plugged with a fungous growth, prolonged so as almost to fill the whole of the left auricle with a mulberry-like tumour.

What is above stated touching the passage of cancerous substance into the veins, applies equally to the *lymphatic system*. Heterologous substance of every kind is taken up by the lymphatic vessels and conveyed to the neighbouring glands in the same manner as pus, (see chap. i,) so that the glands, sometimes alone, sometimes conjointly with the vessels, are plainly implicated in the disease of contiguous parts. In this process the lymphatic vessels are mere passive channels of conveyance, whilst the elaboration of the received substances would seem to take place within the glands. Heterologous products being, for the most part, endowed with the faculty of self-propagation or reproduction, wherever they occur, and being destined, after undergoing a certain number of changes, to cause the degeneration and finally the destruction of implicated textures, scarcely ever become isolated and rendered harmless by assimilation, even in the lymphatic glands; on the contrary, a gland when once engaged and to a certain extent disorganized, offers a fresh starting-point for the disease, which then marches onwards until the whole organism becomes its prey. In this manner the lymphatic glands are often seen to constitute an uninterrupted chain from the cancerous tumour,—conveying morbific matter from one point to another, until it reaches the thoracic duct, and eventually the veins.

Heterologous formations are not all equally subject to the absorbing power of the lymphatic system; indeed the degree of this subjection seems to depend precisely on the degree of their heterogeneous character, while other circumstances, including medical treatment, exercise only a subordinate influence. Thus *tubercular matter,* though very readily taken up and conveyed by the lymphatic vessels to the nearest cluster of glands, seldom passes on to the succeeding group. It will be seen from the statistics in the chapter on Tubercle of the Lungs, that out of 84 cases of pulmonary phthisis the bronchial glands were involved in 21; the mesenteric glands, in 35 cases out of 44 of intestinal tubercle; but that beyond this there was no further diffusion of the tubercular matter. When tubercle originates in the lymphatic glands, as sometimes happens, it never spreads beyond the next series; for instance, from the cervical to beyond the bronchial glands. *Melanosis,* with but rare exceptions, manifests the same relations. On the other hand, heterologous growths susceptible of a higher organization, are far less amenable both in degree and frequency to this species of circumscription; *medullary fungus,* in particular, is not merely transmitted to the nearest glands, but often with extraordinary rapidity from one set to another.

In these cases the lymphatic vessels remain unchanged,—although in the instance of a phthisical female, Andral (Préc, d'Anat. path,) found a partial thickening of the membranes, especially in the neighbourhood of the valves, in the lymphatic vessels situate beneath the serous membrane of a portion of intestine affected with tubercular exulceration. The lacteals leading from intestinal ulcers may often be traced, loaded with tubercular matter, to the adjacent mesenteric glands; and Sömmering frequently found the lymphatics in carcinomatous mammary glands gorged, in like manner, with discoloured ichorous fluid.[1]

The *glands* on the contrary, are essentially altered by the reception of heterologous matter. When the seat of tubercle, they swell moderately, become softer, and imbibe more blood. The morbid substance is disseminated throughout their texture in the form of globules, the size of a pin's head; these gradually accumulate, and ultimately coalesce; and the aggregate constitutes an uniform yellowish white mass, no longer containing the least trace of the natural glandular texture. At this juncture, the gland, especially in young subjects, augments in volume, neighbouring parts are displaced or compressed, and a great deal of mischief arises. The bronchial[1] and mesenteric glands are most subject, both in frequency and in degree, to this species of degeneration; the cervical glands less so, and the axillary and inguinal least of all. After a shorter or longer period, the tubercular mass acting as a foreign substance, excites irritation in the part, and a reaction akin to the inflammatory is set up, which commonly leads to *softening* of the diseased structure. The softening often, though not always, proceeds from the centre of the gland outwards, and pervades it either in totality or in certain portions only. In the latter case, small cavities are found within the hardened texture, filled with a friable cheese-like matter, and with watery fluid. On retrogression of the disease, whether occupying the whole or a part only of the organ, glandular, like pulmonary or other tubercle, is converted into a calcareous mass, sometimes of a pultaceous, sometimes of a dry, mealy or chalky consistency, and sometimes of stony hardness.[2] In the bronchial glands, the tubercular changes above described are frequently accompanied by the infiltration of their texture with a black substance, which some hold to be black pigment of a melanotic nature, others pure carbon.

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[1] De morb. vas. absorb. p. 107.

On the introduction of fungous substances, and especially of medullary fungus, these glands often swell to an extraordinary degree, forming in particular situations, both in the extremities and within the cavities of the body, enormous tumours.[3] In most of these cases they are highly vascular, and undergo ulterior changes in the same order, and under the same circumstances as fungous tumours in the parenchyma of other organs.

For further details, the reader is referred to those sections which treat of diseases of the lungs and of the liver.

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[1] Compare F. G. Becker, Diss. de gland. thor. lymphat. &c. Berol. 1826.

[2] Wallach (Holscher Annalen, vol. ii, fasc. 4) found it to consist for the most part of phosphate of lime and organic matter.

[3] See, for instance, the second figure in Struve's Dis. de fungo pulmon, &c. Lips. 1837.

# CHAPTER V.

## DISEASE OF THE HEART.

### SECT. I.—PERICARDITIS.

*Inflammation of the pericardium,* like that of all serous membranes, is attended with effusion, of variable nature, and susceptible of manifold transformations,—some inevitably fatal in their result,—others again compatible with prolonged life and health.

The opinion was formerly general, that every inflammatory product of serous membranes was originally liquid, and that from this liquid both pus and organized adventitious membranes could be produced. Recent research, however, (as will be seen more fully in the account of pleurisy,) has demonstrated that definite grades of inflammation induce definite forms of exudation, which essentially modify the ulterior course of the disease, and also such changes as the morbid product afterwards undergoes. Lobstein felt the necessity, on other grounds, of assuming several distinctive modifications of inflammation, in relation to its sequelæ, and termed these respectively *epiphlogosis, phlogosis, hyperphlogosis, and metaphlogosis;* assigning to each certain characteristic features, both symptomatic and anatomical, which he has endeavoured to carry out his description of the inflammation of individual organs. It will appear from the following delineation of pericarditis, that inflammatory products do in reality vary in accordance with certain distinctions in the character of the inflammation itself.

The pericardium may become inflamed either throughout, as is probably almost always the case in the more violent grades,—or at certain points only. The so-termed *milk-spots* or islets (insulæ), so often observed on the portion of the pericardium covering the heart, are the remains of partial and slight inflammation. All white patches of the pericardium are not, however, of inflammatory origin, as de la Harpe[1] supposes; nor are they, on the other hand, all referrible, as Hodgkin[2] believes, to mere friction of the surfaces of the sac against each other, or against tumours. The majority of pathologists assume two varieties,—the one—of somewhat rarer occurrence—being decidedly an inflammatory product. Milk spots of this character occupy the free surface of the pericardium, and though cohering by means of blood-vessels, can be easily peeled off without detriment to the serous basement-membrane. They are of various thickness, of exceedingly irregular form and distribution, now sharply defined, now blending almost insensibly with the healthy serous membrane; their surface is sometimes smooth, sometimes rough or villous. They are more frequent at the anterior than at the posterior surface of the heart; most of all, along the course of the coronary vessels, which are spotted, striped, or dotted with them. This variety of milk spot is doubtless met with in persons who had never obviously suffered from thoracic inflammation, and occasionally even in infants. (Hodgkin, l. c.) Nevertheless, it must be looked upon even then as the consequence of inflammatory irritation, although too limited in extent, and too trivial otherwise to have determined any prominent vital symptoms. Many partial adhesions of serous surfaces originate in the same insidious manner. In some instances I have ascertained the inflammatory origin of these milk patches from the co-existence of partial cellular adhesion, and the formation of distinct filamentous bands between the heart and pericardium. These *islets* are, moreover, often found associated with adhesions of the neighbouring pleural and peritoneal surfaces.

*Milk patches* not the result of inflammation, are mere thickenings of the pericardium itself, and not capable of being detached therefrom. They merge insensibly in the healthy structure; according to Bizot,[3] they are always situate upon the right side of the heart, and incomparably more frequent in men than in women; they first appear about the meridian of life, and increase in frequency and compass with advancing age.

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[1] Gaz. méd. 1838. No. 12.

[2] Morbid anatomy of the serous membranes, p. 59.

[3] Recherches sur le cœur, &c. (See Mém. de la Soc. Méd. d'Observation.)

The slighter grades of pericarditis are commonly associated with the effusion of a pale yellowish or reddish serum, seldom quite limpid, but containing a certain proportion of plastic matter, either flocculent, or as a layer investing the pericardial surface more or less, and so thin as often to escape notice. Upon the relative thickness of this membranaceous investment depend the probable chances of the serous fluid being reabsorbed.

Another of the milder forms of pericarditis is productive of an exudation peculiarly prone to become organized; it consists of a yellowish, reddish, or sometimes brownish *jelly*, loosely interposed betwixt the heart and the pericardial sac. This species of inflammatory product often originates in a secondary affection of the pericardium resulting from a pleurisy of both sides, or of the left side only. From the rapid organization of this gelatinous effusion, complete adhesion betwixt the heart and pericardium promptly supervenes, and though lax at first, by degrees becomes firmer and firmer. After a single access of inflammation, the adhesion is equable round the heart; but on any fresh accession, it loosens at certain spots, and in its stead, ecchymosis, or if the inflammation be intense, pus is formed.

Adhesions occurring in the simple manner first described, do not admit of the pericarditis being recognized with certainty during life, as may be gathered by attentively studying the diagnostic signs enumerated by Louis.[1] I observed a case of the kind at the Leipsic hospital, in a man affected with a double pleurisy and with copious effusion on the left side. This patient when convalescent, and able to walk about the ward, was suddenly seized with cerebral apoplexy, and died after a few days. The pericardium was found everywhere united to the heart, by recent, lax, and easily separable cellular adhesions, betwixt which a little reddish serum was infiltrated. Numerous delicate vessels originating from the pericardium were distinctly seen to penetrate the adventitious cellular structure in a parallel course. In this instance, the inflammation of the pericardium had betrayed itself by no distinctive sign; or to speak more correctly, it had been completely masked by the severe pleuritic affection.—Where the pericardial sac becomes the seat of copious serous effusion, of course the converse will happen.

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[1] Mém. Anatomico-Patholog. p. 253.

Where the inflammation sets in with violence, *plastic matter unsusceptible of organization* is thrown out, and the disease mostly becomes chronic. For, although the violence of the inflammation may be eventually broken, its unassimilable product remains an abiding source of irritation; the sustained hyperæmia of the pericardium keeps up a continued secretion from its surface, filling the sac with a dingy reddish and often flaky liquor; meanwhile, the hinderance to the motions of the heart and to the circulation of the blood goes on accumulating, and the patient dies weeks,—perhaps months from the commencement of the disease. The pericardium is then found more or less distended with the above fluid,—often to the amount of a couple of pounds; its free surface coated with a layer of concentrically disposed false membranes, occasionally an inch thick, having a fibrous structure,—which Leo Wolff[1] mistook for muscular,—but without any distinct marks of organization. In other cases the whole pericardial surface is coated with villous, reticulated, and cock's-comb-like membranaceous deposits.[2] In these, notwithstanding the most scrupulous inspection, I have never been able to detect any vestige of organization. Yet the pericardium sometimes appears very vascular, and the assimilative efforts of nature are apparent from the adventitious membrane being here and there numerously marked with red dots; but nowhere is any vascular connexion betwixt it and the pericardium discernible. If, however, ancient records are worthy of credit, even these cases of pericarditis are remediable, as would appear, amongst others, from the well known narrative of the Messenian, Aristomenes with the hairy heart.

The products of pericarditis are not always uniform, even in the same subject, but as before stated, the same exudation frequently contains various elements, which by virtue of the ulterior changes wrought in them, modify the course of the disease in many different ways. This variable composition of the exudation may proceed from alternations in the intensity of the inflammation,—a rapid succession of inflammatory attacks giving rise in turn to serous and sanguineous,—organizable and unorganizable effusion; or else it may proceed from some peculiar ingrained morbid predisposition to engender diseased products. Thus it not unfrequently happens that true tubercle, and even medullary fungus is developed out of the exuded substances, as seen both by Kolletschka and by myself.[1]

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[1] Tractatus Anatom. Pathol. sistens duas observationes in Pericardio. Heidelb. 1832.

[2] Cruveilhier, livr. xvi, pl. ii.

With a view to mark the portentous consequences of a mingling of effusion with the colouring matter of the blood, Laennec distinguished two forms, namely, "*péricardite franche*" and "*péricardite hémorrhagique*;" the latter, of which some very remarkable cases are recorded by Bouillaud, being far less susceptible of cure by absorption, than the simple plastic or serous form. The admixture of *pus* is equally fraught with mischief. It is often detected in the midst of the plastic exudation, and may, on renewal of the attack of violent inflammation, give rise to ichorous, or even to incipient gaseous decomposition of the effused matter. The effusion of mere pus results from the highest degree of inflammation running an impetuous course, and mostly terminating fatally in a few days. The pus is, however, always associated with more or less plastic matter, which is thrown down in the shape of soft yellow flakes, or unorganizable false membrane. It is probable that even pus may, if not exceedingly copious, be sometimes partially absorbed, leaving only its solid parts to undergo ulterior changes about to be described. When, namely, plastic exudation consists partly of organizable, partly unorganizable elements, the former become converted into vascular false membranes and cellular tissue, whilst the latter gradually pass into a whitish or yellowish, soft, cheesy mass, distributed in irregular layers between the heart and pericardium. Into this, calcareous salts are gradually deposited, until it is finally converted into a rough and irregularly shaped earthy concretion. In like manner, a fully organized adventitious membrane may, unless brought by the continued activity of the absorbents to the condition of cellular tissue, take up calcareous materials and form a bony plate.

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[1] See the excellent treatise by Skoda and Kolletschka on Pericarditis, in the Oesterr. Jahrbücher, vol. xxviii, pp. 1 and 2.

The *tubercular* form of pericarditis may occur in two different ways. Either a turbid gray liquor, more or less thin, is at the outset discharged into the sac, whilst the free surfaces of the pericardium become the seat of plastic deposit; that is to say, of an aggregate of grayish white or faint yellowish, flat, and tolerably firm granules, denoting both in form and distribution their tubercular character. Or else a number of grayish white granules, as large as a pin's head, congregate in the midst of plastic effusion, go on accumulating; and, whilst the organizable portion of the effused matter is effecting an adhesion between the heart and pericardium, form into greater or smaller masses of tubercle, which after absorption of the fluid parts, may assume a mealy consistence. In all such cases, tubercles of old or recent date, are found in other organs.[1]

The development of *medullary fungus* within the effusion is very well described by Kolletschka. "The transformation," says he, "is only a gradual one, and at the same period that one portion of the exudation is already in the condition of medullary cancer, an adjacent one is still engaged in the act of transition from the plastic exudation to the fungoid form; whilst a further portion contiguous to the second, betrays no such evidence of degeneration. During the act of medullary transition, the plastic mass becomes paler, white, or light-gray, possesses delicate blood-vessels, and acquires a cellular structure; the cells becoming filled with medullary substance." Medullary cancer is at the same time present in other organs.

Irrespectively of the diverse nature of its product, the pericardium itself exhibits a variety of changes. At first it is always reddened, partly from a bluish red tint diffused in striæ and spots over its surface, partly from the manifest injection of its blood-vessels to their ultimate ramifications. In all cases where organizable, and in most cases where plastic effusion of whatever kind predominates, this character of injection persists during the whole course of the disease. Where, however, the inflammatory product is of a serous or purulent character, it generally happens, after the active inflammation has subsided, that the injection vanishes, and the pericardium appears pale and lack-lustre, and at the same time is somewhat thickened and softened. The cellular texture connecting the pericardium with subjacent parts is often found infiltrated with serum.

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[1] Compare (besides the already cited treatise of Skoda and Kolletschka) Cruveilhier An. Path. livr. xxix.

After the frequent recurrence of inflammation, the pericardium becomes thickened, assumes a semi-cartilaginous hardness, and loses its sero-fibrous character, constituting an almost homogeneous, yellowish-white mass, several lines in thickness.[1] It frequently happens that the fibrous portion alone becomes thickened; probably acquiring thereby a certain aptitude for ossification, which, in a few instances, has been observed in the texture of the pericardium.

The muscular substance of the heart is flaccid, soft and pale, as if macerated and wasted, and that proportionately to the period during which the organ has been exposed to compression from the effused fluid. When, however, the disease has had a short course, it is found darker and of a dingy red. The inner surface of the heart, by virtue of its serous character, is frequently implicated in the inflammation; and mostly found, especially about the auriculo-ventricular valves, tinged dark-red. The ventricles sometimes contain a fibrinous clot deposited in layers, which would seem to have originated during life. In the auricles is found a quantity of dark, semi-coagulated blood, whilst the large venous trunks are gorged with blood. In many cases true endocarditis is present.

The disease is frequently complicated with pleurisy and pneumonia, and not seldom with inflammation of the neighbouring peritoneum; and of the serous covering of the spleen. The lungs are invariably found congested, the inferior lobe frequently adhering to the pleura near the pericardium, but without any further trace of pleurisy being present. In great distension of the pericardium with fluid, the adjacent pulmonary texture is found lax and non-crepitant, as though compressed by pleuritic effusion.

Most of the abdominal organs, and especially the liver, are gorged with blood, as are also the vessels of the brain and of its membranes. The quantity of serum beneath the arachnoid and within the ventricles of the brain is commonly augmented.

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[1] Hohnbaum (Casper's Wochenschrift, 1838, No. 36.) Bidois (Arch. gén. de Méd. vol. iii, 3me série, p. 511.)

The subcutaneous cellular texture is universally, though more particularly in the lower extremities, prone to serous infiltration, and that in a degree proportionate to the duration of the pericarditis and to the amount of liquid effusion.

Acute rheumatism of the joints, (it may be with inflammatory exudation in the capsular membranes) frequently precedes or accompanies pericarditis. The serious nature of this complication was first clearly shown by Bouillaud.[1]

In 72 cases, noted by Hache[2] and others, there was concurrent articular rheumatism sixteen times, being almost a fourth of the whole number.

Young persons seem chiefly prone to pericarditis. Out of 55 cases, 6 only referred to individuals beyond the fortieth year. In males again, the predisposition would appear to exceed that in females, in the proportion of four to one. (Hache.)

## SECT. II.—CARDITIS.

Most pathologists distinguish three forms of inflammation corresponding to the three textures of which the heart is made up, namely, inflammation of the serous investment, which, upon the whole, presents the same anatomical character as that of other serous membranes, and has been discussed in the preceding chapter; secondly, inflammation of the muscular substance of the heart, which differs from that of muscle generally only in as far as its function and the disposition of its fibrous layers are peculiar; and, finally, endocarditis or inflammation of the lining membrane of the heart, the analogy of which to common inflammation of the blood-vessels is obvious. If the relation between the affection of these several textures and that of kindred structures elsewhere be brought to bear upon individual cases, according to the principles of general anatomy, it will be found less difficult to form an opinion respecting them, and more easy to avoid the numerous errors into which earlier pathologists have fallen. For, even recently, various changes affecting the heart's substance have been mentioned under the head of inflammation, though strictly speaking quite unconnected with it. Laennec's critical remarks established a more correct standard in this respect. Nevertheless the term *carditis,* and more especially *endocarditis* has been employed by Bouillaud in too vague a manner.

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[1] [The connexion between articular rheumatism and pericarditis was distinctly pointed out by Dr. P. M. Latham, in the course of his Lectures, published in the third volume of the 'London Medical Gazette,' six years prior to the appearance of M. Bouillaud's work.—From a recent publication of Dr. Latham's, however, it will be seen that acute rheumatism is far more frequently complicated with endocarditis alone, than with pericarditis alone, or than with the two concurrently. (See Lectures on Diseases of the Heart. London, 1845. Lecture VIII.)—Ed.]

[2] Arch. gén. 2me. sér. vol. ix, p. 326.

It has been matter of doubt whether instances really have occurred of *general* carditis, that is to say, where all three forms have coexisted. Laennec admits only a single authentic case of the kind, namely, the oft-cited one of the elder Meckel,[1] ample details of which are given by Kreysig. I am acquainted with but a single example of the same character of a more recent date, namely, that recorded by Th. Salter,[2] in which pericarditis was associated with purulent softening of the muscular substance, more particularly of the left chambers, and with fibrinous deposits, probably of inflammatory origin, within the cavities of the heart. But however rare an instance of *intense* general carditis, the coincidence of the three forms in a minor degree is sufficiently common; thus in endocarditis certain portions of the muscular substance and likewise of the pericardium are frequently found more or less affected, or conversely. Upon the whole it may be assumed that no one of the forms can occur in a high degree without more or less implicating the other textures of the heart. The frequent concurrence of pericarditis with endocarditis has been demonstrated by Bouillaud. We must, however, guard against confounding with carditis those cases of pericarditis and of pleurisy of the left side, in which the substance of the heart is either flaccid, pale, and softened, or here and there dark coloured and pulpy. As well might a diaphragm softened and discoloured by inflammation of the superincumbent pleura be reckoned as inflamed. In both instances, indeed, the influence of the neighbouring inflammatory process is too obvious to be called in question.

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[1] Mém. de l'Acad. de Berlin. Ann. 1756, p. 31.

[2] Lond. Med. Gazette, Jan. 1839, p. 618. [To these cases might be added that of Mr. Stanley. See Medico-Chir. Trans. 1816. A well marked instance of general carditis is detailed in Dr. J. R. Bennett's 'Report on the Progress of Pathology,' &c., (Brit. and For. Med. Rev., No. xxxix.,) as taken from the Bulletin de l'Acad. Roy. de Méd. April 1843.—Ed.]

Although partial *myocarditis* is by no means rare, true idiopathic inflammation of the *whole* cardiac muscle is exceedingly so; only a few isolated instances are upon record. According to the leading authorities on disease of the heart (Burns, Kreysig, Laennec, Hope, Bouillaud,) inflammation is productive of the following changes in the muscular substance of this organ: serous infiltration into the cellular texture uniting the muscular fibres; sero-lardaceous degeneration of the same texture with red, brown, or grayish-white disorganization of the muscular fibres themselves; purulent infiltration of the cardiac muscle, or yellow and grayish softening; finally, abscess. Although Bouillaud and others refer ulcer of the heart, aneurism, induration, and ossification within the muscular substance to the same cause, it appears more expedient to treat of them in the sequel, as they depend on other morbid processes besides inflammation.

The first trace of inflammatory action in the heart's muscle is an injected condition of the minute vessels, which in a dense network traverse the cellular texture situate between the fibres, the heart's walls being darker coloured and firmer than natural. Presently, however, the cellular texture becomes infiltrated with serum, either clear, or turbid,—either thin and watery, or of the consistency of plastic lymph. Occasionally this exudation is saturated with the colouring matter of the blood, and occasionally it is mingled with fat-globules. The muscular fibres lose their consistence through maceration in this their degenerate nutrient web; they lie in it as it were isolated, and assume, according to the peculiarity of the inflammatory product, a pale, dingy-gray,—a yellowish,—or a dirty-brown hue. The whole mass is flabby, friable, and easily compressible into a pulp. By and by the effusion assumes a lardaceous character, and the parts involved, a homogeneous and whitish aspect; or else the exuded matter, first becoming of the consistency of boiled white of egg, and of the colour of pus, (Laennec,) rapidly undergoes purulent softening. Up to this point, as Gendrin[1] imagined, and Gluge[2] has since shown, the primitive fibres of the muscle preserve their peculiar texture and form, but directly afterwards share in the subsequent change and destruction of the part.

*Suppuration* is either diffuse, so that, within a space not well defined, the fibres—partially loosened and released from their continuity—lie bathed in the purulent liquor as if softened by maceration, and are reducible by the slightest compression, into a shapeless pulp; or else the pus collects in circumscribed masses, and one or more abscesses, of various size, form within the walls of the ventricles and auricles, (Bouillaud,) pointing, for the most part, outwardly, and causing a bulging of the simultaneously inflamed pericardium. The purulent matter is not invariably yellow, but sometimes, in the first instance, of a more or less brownish tinge, owing to admixture with the colouring principle of the blood. These abscesses, resulting from carditis, ought to be carefully distinguished from the purulent depôts produced in the substance of the heart, as in other organs, by phlebitis,[3]—not unknown even to Morgagni.[4]

How far such partial or general softening of the heart's walls may lead to *rupture* and to an influx of blood into the pericardium will be discussed hereafter. Walther, a pathologist of a former day, has furnished one example of this nature. It will be shown in the sequel, that true aneurisms of the heart are, in the majority of cases, dependent upon muscular carditis.

Whether *gangrene* occurs in the heart is still undetermined; Laënnec and Bouillaud[5] will not entertain any of the cases adduced. On the other hand, Kreysig and Lobstein[6] cite instances from Portal and others, in which, after disease had run a hurried and violent course, the substance of the heart was found softened in irregular stripes and patches of a dark blue colour approaching to black, and emitted a fetid, distinctly gangrenous smell. In this last circumstance alone does it differ from what Cruveilhier[1] has described as *apoplexy of the heart,* which consists in partial softening with infiltration of dark blood between the muscular fibres, and eventual perforation of the heart's walls. An apoplexy of the heart of this kind, unattended with rupture, is instanced by Kreysig,[2] among his cases of carditis.

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[1] Gendrin, (loc. cit.)

[2] Gluge (Mikrosk. Unters. fasc. i, p. 33.)

[3] Carswell, fasc. pus. pl. i, fig. 5.

[4] Epist. xxv, art. 20.

[5] Mémoires de Berlin, 1785.

[6] Anat. Path. vol. ii, p. 490.

Myocarditis generally attacks the left side of the heart, and most frequently the ventricle. This is more especially the case in that *partial* form which accompanies endocarditis, and in which isolated points or patches of the innermost muscular layer, of the size of a pin's head and upwards, are found altered by inflammatory effusion. This alteration appears at first to consist in a maroon coloured infiltration, which presently changes to yellow, and ultimately almost to white, and may be quite even and flat, or else throw the internal surface of the heart slightly into relief. The portions of muscle thus disorganized gradually wither away, a thing particularly common in the papillary muscles, or they merge into little whitish yellow knots, cords or callosities, which long continue to denote the previous inflammation. I had once an opportunity of examining, by the microscope, the inflamed muscular texture in the state of white softening above mentioned, and detected a very small quantity of amorphous, grayish effusion between the muscular fibres. These latter I could distinctly trace into the inflamed part, and I could at the same time perceive that the transverse fibres were lost at the edge of the aforesaid part, the longitudinal ones being somewhat thickened, as if œdematous, and ultimately resolved into a vast number of very delicate fibrils, resembling in aspect and in their undulating course, the fibres of cellular tissue.

Myocarditis is not confined to any particular age. Its rare occurrence prevents any *positive* inference being drawn as to its relative frequency in the two sexes. Out of twelve authentic cases given by various authors, nine were males and only three females.

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[1] Livr. iii, pl. i, fig. 1, and livr. xxii, pl. iii, fig. 1; Albers, (Atlas. Abth. iii, vol. xi *a*, fig. 2.)

[2] Herz Krankheiten, vol. ii, sect. i, p. 272.

## SECT. III.—ENDOCARDITIS.

*Endocarditis*[1] is far more frequent than muscular carditis, although the latitude assigned by Bouillaud to the former needs limitation. It is very difficult to delineate its anatomical features with such truth and precision as to enable them to be definitely recognized in every case, and we shall have a better chance of rendering them intelligible by negative than by positive means. It will, however, be well to premise, that in considering the internal investment of the heart and the general lining membrane of blood-vessels, too much stress must not be laid on their alleged analogy with the serous membranes. The former are not only otherwise circumstanced in being exposed to the perpetual current of blood, but differ materially even in structure from true serous membranes.

Reddening of the endocardium is spoken of as decided evidence of inflammation. According to Bouillaud it is of various degrees of intensity, sometimes of a rose-colour, sometimes scarlet, sometimes violet, or of a deep, occasionally even of a brown red; and it may be either partial or general. All these tints are, in reality, only modifications of scarlet and violet, and may here result from a great variety of circumstances besides inflammation. The shades of violet coloration are, in most instances, produced by typhoid decomposition of the blood. I have frequently observed them in variable degrees of intensity in typhous subjects, twelve or sixteen hours after death, when the temperature of the atmosphere has been very low,—as also where the blood has become vitiated through phlebitis, and generally after the absorption of pus,—for example in phthisis. In such cases the substance of the heart is, at the same time, found pale and flaccid, whilst the pericardial sac sometimes contains a thin, dingy red fluid. The deep red, or brown-red coloration will, on the other hand, be apparent in those cases of pneumonia, formerly denominated typhoid, in which, owing to the great extent of lung involved, the decarbonization of the blood is extremely defective, and the diseased fluid has time to exercise its manifold influence upon the nervous system and the rest of the frame. A similar reddening is met with in the bodies of persons who have died with signs of decomposition of the blood, brought on by the abuse of spirituous liquors. In all these the morbid stain, for obvious reasons, varies in intensity, and cannot be distinguished from that of inflammation, either by ablution or by maceration. Finally, the scarlet hue, as observed in the lining membrane of the heart and blood-vessels, may at any time be produced by briefly exposing the parts, even if perfectly healthy, to the action of the air. From all this may be inferred the great difficulty of ascertaining the real source of discoloration. Indeed the examples of reddening without inflammation are of the most common occurrence, and many more might be appended to the above, did space permit. With respect to the circumstances under which the peculiar *cadaveric reddening* occurs, satisfactory information may be gathered from the interesting experiments of Trousseau and Rigot.[1]

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[1] Bouillaud arrogates to himself the merit of having first directed attention to this disease. If, however, he had read Kreysig's work, he would have there found almost everything he has written on endocarditis, due allowance being made for the less advanced state of anatomical pathology of that day. Kreysig does not, it is true, award to the disease so vast a field as Bouillaud, but perhaps no one will blame him for this. My own descriptions will be found to differ in many particulars from those of Bouillaud. The best anatomical account of endocarditis of modern date is that of Rokitansky, (Handbuch der Pathol. Anat. vol. ii, p. 425, &c.) to which, in the ensuing chapters, we shall repeatedly refer.

A product of inflammatory action deserving of notice here, are the *fibrinous coagula* occurring within the cavities of the heart. Formerly much importance was attached to these so-termed polypi of the heart, and no small disposition was evinced to regard them, not only as the immediate agent of death, but likewise as the main source of many chronic diseases of the respiratory and circulating organs. Pasta[2] combated this extravagant notion, but went too far in asserting that every polypoid formation within the heart was the result of stagnation of blood after death, and that even fibrinous deposition within aneurismal sacs was impossible during life. Afterwards, when organic diseases of the heart were more minutely studied, fibrinous coagula became the subject of repeated research, and it is Kreysig's merit to have shown that, although they could not be looked upon as either the cause or the product of chronic disease, they might yet be the result of an inflammatory process affecting the inner surface of the heart; in short, that a polypous carditis must be recognized. Bouillaud arrived at the same conclusions, but, on the other hand, perhaps assigned undue weight to endocarditis in the production of polypi.

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[1] Arch. gén. 1826, and June 1827.

[2] Pasta, Joseph, (de Sanguine atque de Sanguinis concretionibus. Bergami, 1786.)

Of the blood coagula, two types are commonly acknowledged, the distinctions between which are essential and obvious; there are, however, so many transition-forms, that it is often extremely difficult to come to a definite conclusion.[1] The first type is characterized by the simple fibrinous clot, so common in robust individuals. It is yellow, pellucid, of the elasticity of jelly, of a smooth shining surface, and fashioned to the shape of the heart's cavities, especially at the origin of the pulmonary artery, where it often appears moulded with a faint impression of the valves. It seldom fills the cavities however, and never adheres to their parietes. This gelatino-fibrinous mass occasionally covers a clot of coagulated blood, in the same proportion as the buffy coat the crassamentum in blood drawn from a vein; it, however, frequently constitutes the bulk of the concrement, and contains only a few portions striated or dotted blood-red. Polypi of this description, often proceeding with flattened ends into the large vessels, and being usually of greater volume in the right half of the heart than in the left, are developed either during the agony, or after death; but result, in no instance, from disease of the endocardium. The second type of polypoid coagula is characterized by opaque, white or dingy-gray, soft, filamentous and elastic concrements, consisting of several irregularly superimposed layers, with an uneven surface more or less firmly adherent, especially in the neighbourhood of the valves, to the heart's parietes, and insinuating their fangs among the columnæ carneæ and tendinous cords. They rarely form a single coherent mass like the foregoing species, but rather unequal and distinct accumulations, connected together through the medium of coagulated blood, or else of a brown or gray pultaceous substance. In rare instances, several of the fibrinous layers include in their centre purulent fluid. At their surface, and even between the different layers, dots and little stripes of blood are discernible. The majority of these coagula are developed during life, and are frequently the *immediate cause* of death. Bouillaud regards them as products of an inflammatory process affecting the endocardium, and as being partly secreted by this membrane, partly deposited by the blood while passing over the inflamed parts.

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[1] Concerning the microscopic and chemical relations of coagulate fibrin, see Henle's Annual Report, in Henle and Pfeuffer's Journ. 1843, p. 168.

An unbiassed survey of the cases in which these polypi occur, will render it apparent that their development does not necessarily depend on inflammation of the endocardium, but that the seat of the morbid action is often, nay, in the majority of instances, remote from the heart. In proving this, the fact should be borne in mind, that certain substances when brought in contact with the blood cause its coagulation, and this the more rapidly, the larger the amount of blood experimented upon, and the more briskly they are agitated and mingled with it. According to the experience of most pathologists, such substances are met with in the blood itself,—tubercle, for instance, cancerous matter, and more frequently pus, which last is particularly efficient in promoting coagulation. These entering the circulation by degrees, will not fail, at whatever part of the body engendered, to accumulate eventually within the cavities of the heart. For it is well known that the ventricles do not empty themselves completely during systole, but retain a small portion of blood; this, being by the shock of the heart's contraction impelled into the network of the columnæ carneæ, acquires there, under the circumstances above indicated, the opportunity to coagulate; the same relations subsisting, the coagula, at first small, gradually increase in bulk, so as eventually to impede, and ultimately to obstruct the circulation, and after death to present a fibrinous concrement of the character last described. It follows from the foregoing explanation, that even those polypi which contain fluid pus, are not necessarily the product of endocarditis.[1]

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[1] Cruveilhier (Anat. Path. livr. 25) expresses himself, with regard to the appearance of pus in the coagula, as follows: 1st. The pus is generated at a distance from the heart, is carried thither with the circulating current, and there invested with a

At all events, polypi of the heart bearing all the indications of having originated during life, and where there is no warrant for supposing concurrent endocarditis, are found in most fatal instances of phlebitis (see the article), of pneumonia in the third stage, of the absorption of pus in extensive sores, and frequently even in the instance of softening tubercle of the lung. Several of Bouillaud's cases probably belong to this category.

It would lead to endless confusion were the endocardium in its relation to morbid products, to be viewed as the analogue of serous membranes. For the products of these latter when inflamed, are most heterogeneous,—varying from gelatinous effusion to liquid pus; so that, judging by such analogy, it would be impossible to say whether the polypous coagulum arose from endocarditis or not. A more correct and available comparison might be instituted with inflamed blood-vessels.

Having thus greatly curtailed the value of the two great distinctive features of endocarditis, we shall next proceed to point out the means to discriminate between redness of the endocardium from inflammation, and redness from imbibition; as also between polypous formations engendered by exudation, and coagula resulting from the inflammatory irritation of other organs.

The inflammatory redness is almost always spotted,[1]—pale and dark alternately; in one place more of a violet, in another more of a scarlet colour. The redness of imbibition is, on the contrary, more equable throughout, and darker, perhaps, where the blood makes a prolonged sojourn. The redness of imbibition is therefore almost invariably observed in the following descending order: darkest in the right auricle, paler in the right ventricle, with the exception of the valves of the pulmonary artery, which are as deeply coloured as the auricle;

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fibrinous capsule. 2d. The pus is formed within the heart, either by a chemical conversion of the coagulum itself, or by an inflammatory process within the latter, (the opinion of Dupuytren and Legroux,) or, lastly, by inflammation of the endocardium. Cruveilhier conceives that the pus thus formed reaches the centre of the coagulum through capillary action. It would be more simple to assume that the coagulation of the blood is the consequence of the presence of pus, since the latter is wont always to occur within a fibrinous envelope.

[1] It is doubtful whether the spotted redness occurring on the inner surface of the heart in persons poisoned with corrosive sublimate is of an inflammatory nature or not.

still paler in the left auricle, whilst the left ventricle often retains quite its natural tint, except that the aortal valves are darker; in the great vessels the posterior surface is strikingly dark in comparison with the anterior. Moreover, the endocardium continues smooth and polished, and in all other respects natural, whilst inflamed portions of it assume a dull, velvety appearance, and are sometimes thickened and softened. If besides the polypous coagulum the endocardial surface is invested with a soft, firmly adherent false membrane, the inflammatory state is no longer doubtful. At the valves, particularly the tricuspid and mitral, this membranaceous covering takes the character of soft, hemispherical granulations, capable of increasing in bulk, and in some instances, as shown by Kreysig and Bouillaud, susceptible of organization. Secondly, these polypoid concretions can be regarded as products of endocarditis only when met with on the inner surface of the heart, in the manner before described, or when they cannot be traced to any other morbific condition. Here absence of inflammatory redness is not always a sufficient reason for denying their inflammatory origin,—seeing that the redness is very fugitive, more particularly in intense degrees of inflammation, when the colour of the membrane gradually from a dull and equable brown red becomes paler,—changing to a dirty grayish yellow. Carswell[1] has figured a remarkable case of inflammatory polypus, in which the lining of the heart appears preternaturally pale. This polypus was partly of a grayish, partly of a lively red colour; it exhibited both on its surface and in its substance little drops and stripes of pus, had insinuated itself amongst the columnæ carneæ, and adhered as if glued to the equally pallid mitral valve. Legroux and others contend that the pus is the result of inflammation of the fibrinous layers, within which it is sometimes found to accumulate,—an error palpably refuted by Bouillaud. Admitting the above granulations resulting from exudation to be susceptible of organization, it may still be doubted whether the polypous coagula are so. Otto could never detect in them any vascular development. It is true that they exhibit points and streaks of blood, sometimes extending into their interior, and which might certainly *pass for* the rudiments of organization; but it is not conceivable that foreign bodies of such dimensions could occupy the central organ of circulation without speedily inducing death. The few authenticated instances of organized polypi leave it problematical whether they were anything more than mere vegetations upon the valves; and vegetations, there as elsewhere, can hardly be reckoned among the immediate products of inflammation.

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[1] Fasc. viii, pl. iii, fig. 7.

The first and most constant change resulting from endocarditis relates to the endocardial membrane. This, after the aforesaid red spots and patches have disappeared, loses its smooth pellucid aspect, becomes relaxed, turgescent, and rough, puts on a grayish and dull appearance, and is capable of being stripped off with comparative ease. Rokitansky states that under these circumstances the endocardium frequently becomes the seat of irregularly-jagged fissures, which forthwith form a depot for a grayish yellow coagulum.

When the inflammation assails more particularly the apparatus of the auricular valves, a still more frequent occurrence is the rupture of one or more of the papillary tendons. This is particularly common at the mitral valves. The filaments or shreds of such lacerated tendons curl up and form little elevations, invested with fibrinous coagulum and hardened lymph, whilst the valve, previously angular, recedes and acquires a broad raised margin.

In a few instances I have found the semilunar valves both of the aorta and of the pulmonary artery softened and torn into shreds and filaments, which, covered with little wedge-shaped pellets of coagulum and effused matter, floated in the arterial tube in the direction of the blood current.

Bouillaud assumes three distinct stages in the development of the organic changes incidental to endocarditis. In the first stage, reddening and softening of the endocardium unite with the formation of adventitious membranes, fibrinous coagula, and pus. In the second stage these latter acquire form and consistency, begin to become organized and to consolidate either into hard, elevated yellowish, or milk-white spots, or else into granulations partly isolated, partly grouped together; or there may be thickening and shortening of the tendinous fibres with spotted, wrinkled induration of the valves, or, lastly, adhesion of the latter to each other or to the parietes of the heart. During a third stage these products acquire stability, pass into a true cartilaginous or bony condition, and thereby induce coarctation,—but in some instances widening of the various orifices of the heart. Thus all defects of the valves, all ossifications,—in a word, all disease of the heart's apertures,—are directly ascribed to endocarditis. This view does not, however, fully correspond with facts; for, in the first place, experience shows that the several organic changes above alluded to, affect the left side of the heart far more frequently than the right; whilst, according to Bouillaud, endocarditis is observed with equal frequency on both sides, and with most intensity on the right. In the second place we meet with those indurations and ossifications, especially after the prime of life, too often to admit their having been universally preceded by endocarditis; in many subjects, indeed, the contrary could be proved. Thirdly, most of those changes present, in the mode of their development, so perfect a similarity to the progress of atheromatous disease of the arteries as to render it more appropriate to class them under this head.

It cannot be denied, however, that, under propitious circumstances, endocarditis may be productive of thickening of the internal lining of the heart and of the valves, as well as of granulations and ossifications of the latter,[1] though much less frequently than Bouillaud pretends. In fact, ossification, even when met with in young individuals, cannot invariably be ascribed to endocarditis. After careful consideration of all the circumstances, it would appear that the following organic changes arise from either a partial or a general inflammation of the internal surface of the heart: the milk-white or mother-of-pearl coloured patches and thickenings,—perhaps themselves the relics of earlier relaxation, disorganization, and scar-like puckerings,—of the endocardium; secondly, membranaceous deposits on the inner surface of the heart, more especially in the left auricle, and also in the left ventricle, upon the septum and about the origin of the aorta. These have the appearance of elevated, rough, and puckered connecting membranes. They are of a dull yellow tint, and are easily separated. To the same class belong adhesions of the valves amongst each other, and with the heart's parietes, rupture of the tendinous filaments of the auricular valves; and, finally, a state of partial destruction of the valves, with irregular notchings, and fringed appendices of variable length. This has heretofore been observed only in the semilunar valves of the aorta, and twice by myself in the pulmonary artery. Sometimes it is complicated with partial adhesion of the valves together.

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[1] Such granular ossifications are figured in Albers' Atlas, sect. iii, pl. iii, fig. 4.

These results of inflammation, the spots on the endocardium alone excepted, are by no means of trifling import, inasmuch as they lay the foundation for secondary organic disease of the heart, and by narrowing or dilating the heart's orifices, for hypertrophy and dilatation of the implicated ventricles and auricles. In this point of view, individual cases of these affections are to be considered as really connected with endocarditis. Dilatation of the implicated cavities not unfrequently originates in inflammation of the inner surface of the heart, though still less rarely in a complication of endocarditis with pericarditis. The dilatation is then owing to the muscular layers of the heart being rendered lax, and in a certain degree paralysed by the neighbouring inflammation, so that their contractions become weaker, and blood accumulates within them. *Adhesions of the valves* exert an especial influence in relation to consecutive diseases of the heart. Bouillaud has described many examples of the kind, and Albers, also, in his Atlas;[1] they appear, upon the whole, not to be uncommon, and will assuredly be met with at times by persons who have frequent opportunity of examining bodies after death. *The semilunar valves of the aorta* are the most frequent seat of these adhesions, although the pulmonary artery is by no means exempt. All three valves may cohere at their margins in such wise as to leave but a very small opening of variable form, for the passage of the blood.

More frequently, however, two of them only coalesce,—these, generally the two opposed to the origin of the coronary arteries. The adhesion commences at the common point of attachment of two valves, and thence spreads to the centre of their free margin, reversing their shape and direction, so that they present a concave surface to the ventricle and a convex to the aorta. When they preserve their pouch-like character, the orifice of the artery becomes contracted; when, on the contrary, they adhere simultaneously to the parietes of the artery, the mouth of the latter may become permanently dilated; this effect being further promoted by the circumstance that the valves are no longer capable of preventing the regurgitation of blood during diastole. This last evil may, however, exist, even when the orifice is permanently contracted, since the conjunct valves mostly assume a cartilaginous hardness, which unfits them for entire closure of the orifice.

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[1] Section iii, pl. iv, fig. 2, serving at the same time as an example of simultaneous pericarditis and endocarditis, and plate vii, fig. 1.

*Cohesion* of the *auricular valves* is far more rare. Laennec met with one instance of it in both auricular valves, as well as in those of the aorta, but regarded it as congenital. Bouillaud has seen it four times in the mitral, and once in the tricuspid valve. The necessary consequence is, abiding dilatation or else contraction of the ostium venosum. It is still more rare to discover papillary muscles adhering together or to the walls of the heart; the tendinous filaments are, however, frequently found united into cordlike bundles.

Finally, we have to advert to the secondary phenomena often produced by endocarditis. They are referrible to the effusion almost always consequent upon that affection, in the various forms and localities already specified. A considerable portion of the effused substances being carried away in the first instance by the circulation, and another portion during the subsequent period of softening, it is obvious that these inflammatory products must, just as in phlebitis, act a subordinate part within the capillary system. The *spleen* and *kidneys* appear particularly liable to such changes. In the spleen coagulated fibrin is found of some breadth at the periphery, but gradually tapering towards the centre, having mostly a tolerably sharp outline and a brownish-yellow hue. Smaller deposits, of a similar kind, are met with in the cortical substance of the kidney; sometimes even intersecting the papillæ. According as the inflammatory product of the endocardium partakes more of the character of fibrin or of pus, the secondary deposit will be of a more or less consistent kind, and shrivel in the event of recovery,—or else liquefy, and terminate in abscess. Though frequent in the localities above indicated, these secondary phenomena are proportionately rare in other organs, as in the liver, for instance. Sometimes, however, they occur in the serous cavities. Endocardial inflammation may occupy the whole inner surface of one side of the heart, and in rare instances of both sides; although mostly limited to isolated portions, or to particular valves. It most frequently affects the left side of the heart and the mitral valve. It chiefly attacks male adults from the 20th to the 35th year.

*Fœtal endocarditis.* Rokitansky is of opinion that endocarditis sometimes occurs in the fœtus. Here the disease would probably be seated in the right side of the heart, where it would serve partly to arrest development, partly to prevent the obliteration of the foramen ovale and of Botalli's duct, by directly causing coarctation or closure of the origin of the pulmonary artery. The preternatural conditions which give rise to cyanosis, and which have been set down as simple vices of organization, may possibly, in many instances, depend upon inflammation of the fœtal endocardium.

### SECT. IV.—THICKENING AND OSSIFICATION OF THE ENDOCARDIUM AND VALVES, ULCERS, ANEURISM, AND PERFORATION OF THE HEART—CONSEQUENT UPON INFLAMMATORY AND ATHEROMATOUS DISEASE.

All writers agree as to the chronic nature of the above affections, but by no means as to their real import. Kreysig was disposed to refer them to an inflammatory origin; Bouillaud unhesitatingly ascribes them either to carditis, or to endocarditis; whilst others (Laennec, Andral, and Lobstein) have classed them under various heads. With regard to thickening and ossification of the valves, and of the endocardium generally, it has already been noticed why they are only in certain cases directly dependent on inflammation. The same will apply to ulcer and consecutive perforation, as well as to the so-termed partial or true aneurism of the heart. Lobstein traces most of these morbid conditions to gout as their source, and the evidence he adduces is in many instances most convincing; still it would be restricting those organic changes within too narrow limits to ascribe them solely to the gouty diathesis. We shall proceed to consider them, first, in their analogy to disease of the arteries, which often coexists with them in the form of atheromatous degeneration; and, secondly, as sequelæ of inflammation, especially of the endocardium, and of the substance of the heart.

Thickening and ossification are exceedingly frequent at the different valves, but very rare in other parts of the heart's internal surface. They begin with a dull appearance of the endocardium, at first scarcely discernible, but soon rendered more conspicuous, and associated with a grayish or yellowish tint, the membrane becoming progressively thicker and thicker. Beneath these dull and thickened spots atheromatous matter is deposited, but in slight quantity, and almost exclusively at the valves. The transition to ossification is very rapid, the calcareous deposit, in rough granules, taking the place of the small raised yellow spot, before the latter has had time to spread; so that, instead of the flat scaly ossifications met with in the aorta, we have here rough, tuberous, calcareous masses, presenting jagged, uneven, thick laminæ, or granular nodules of unequal size. They are for the most part of firm cohesion and stony hardness; sometimes, however, of a loose and friable texture; or they conceal beneath a firm crust a pulverulent, or pap-like mass. In the two latter instances it is often difficult, and requires a careful inquiry into all the collateral circumstances, to decide whether these calcareous concrements originate from atheroma, or from endocarditis.

*The mitral valve* is that most subject to atheromatous changes, particularly the portion of its attached margin, situate on the auricular side. Frequently, however, both divisions of this valve are so completely ossified, as to leave but a very narrow chink of communication between the auricle and the ventricle. In other instances isolated, tuberous, and jagged calcareous deposits at the base of the valve encircle the mouth of the auricle, and sometimes spread far downwards, so that with the simultaneous adhesion of the margins of the valves, there remains merely a roundish orifice, the size of a quill. *The semilunar valves of the aorta* are almost in equal degree subject to ossifications, irregularly prominent like the above, and situate at the free margins,—more especially at the sesamoid tubercles. From thence they gradually spread, so as, in some cases, eventually to occupy the surface of one, or even of all the valves. These, meanwhile, frequently coalesce at their margins, so as ultimately to constitute an irregular projecting ring. Hereby the mouth of the aorta becomes naturally more or less narrowed, and is sometimes reduced to a small irregular triangular orifice. Valves thus indurated are capable of offering but a very faint resistance to the backward flow of the blood, during diastole. When the mitral and semilunar valves are altered at their base in the manner described, the ossification, in some instances, extends to the endocardium, in the direction of the *transverse groove* of the heart, constituting an imperfect circular border around the point of union between the walls of the auricles and ventricles.

In other situations atheromatous and calcareous depositions form beneath the endocardium,[1] but more seldom; nevertheless a trifling amount of calcareous substance is often met with in the interior of the tendinous cords, and on the papillary muscles. They are obviously developed at the apex of the papillary muscles from a yellowish substance, moderately hard, and resembling boiled white of egg, which accumulates beneath the thickened endocardium. This displaces the muscular fibres, and thereby gives to the ends of the papillary muscles an angular, abruptly acuminated appearance, instead of their natural taper form.

It is questionable, however, whether those extensive ossifications seen by Haller, Renauldin,[2] and others, within the muscular substance have a similar origin. Perhaps they are derived from inflammation, in the same manner as the calcareous incrustations, described as the sequence of pericarditis, on the surface of the heart. Of this a striking example is given in Baillie's 'Series of Engravings.' In reference to these latter concretions, the following remarks of Kolletschka, founded upon facts, are deserving of notice. He says: "they interfere with the nutrition of the parts above and below, occasion retiform perforation of the pericardium, absorption of the substance of the heart, and, progressively accumulating, penetrate deeply into the muscle, so as, in some few instances, to displace a portion of the heart's parietes, and to project into the ventricle."[1]

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[1] See cases in Voigtel, vol. i, p. 431.

[2] In a male subject, aged 33, he found the walls of the left ventricle petrified, as it were, being in some places granular, and in others crystalline. The fleshy columns were petrified and enlarged, and, without having changed their form, resembled stalactites.

We frequently meet with more or less extensive induration of the endocardium, (independently of ossification,) which affects by preference the valves, and especially the tendinous filaments of the mitral valve. This valve is thickened throughout, but more particularly at its free edges, and is of an equable, pale yellow hue. Its elasticity is lost, and its original texture scarcely cognizable, the whole being swollen into a homogeneous mass, presenting on incision a smooth shining surface. The filaments become thickened in a still higher, probably a six-fold degree, and lose their tendinous character; their external investment assuming a dull gray aspect, and an almost cartilaginous firmness, whilst a deep-red, nearly homogeneous mass is contained within. This degeneration proceeds to involve partly the mitral valve itself, giving it the semblance of a flat, shapeless mass,—partly also the papillary muscles, converting their fibrous texture into the above dark red homogeneous substance. At the same time the tendinous portions are considerably shortened, and the whole constitutes a thick fleshy web of semi-cartilaginous hardness. This high pitch of degeneration is just as rare as slight thickening and induration, especially at the base of the semi-lunar valves of the aorta, are common. The extensive induration above described is always a result of endocarditis of variable intensity.

An inflammatory origin cannot, however, be assigned to every instance of thickening and puckering of the internal surface of the heart. It must here be repeated, that sufficient attention can hardly be paid to the slighter grades,—the first traces of organic changes within the heart,—and to their gradual development; for assuredly the one-sidedness and discrepancy of opinion which prevail respecting their proper nature, are solely chargeable to the practice of limiting minute anatomical inquiry to such cases as have excited interest by marked symptoms during life. It is, however, known that when organic affections manifest distinct vital phenomena, they are far beyond the period of primal development. Hence anatomical details, apparently insignificant, acquire weight when properly linked together. On this score the researches of Bizot, who accurately examined the condition of the heart and arteries in 158 subjects of different ages and sex, (who had *not* suffered from confirmed disease of the heart,) are most instructive. So far as I have been able to put his statements to the proof, I find them, with some few exceptions, quite correct. Bizot noted the first stages of these degenerations in young subjects at certain situations within the heart, and found that they progressively increased in frequency and importance with advancing age. They were met with oftener, and at an earlier period in males than in females. These statistics correspond entirely with those of atheromatous disease in the arteries, with which they present another marked point of analogy, namely, in their almost exclusively affecting the *arterial* cavities of the heart,—the right side only when there exists a preternatural communication between the two divisions of the heart. When discovered in younger individuals in the advanced stage, in which, *as a general rule*, they belong only to the aged, the exception is referrible to marked predisposition, or to the repeated influence of occasional causes.[1] It might appear, from the above, that the organic changes described were a necessary or physiological attribute of old age; a cursory glance at Bizot's tables would, however, show that such is not the fact; if indeed the daily experience of most pathologists did not demonstrate that the heart, even in very old people, is often quite exempt from all morbid products. There is no good evidence of the development of *ulcers* from atheromatous deposits within the cavities of the heart, however frequent in the arteries; they have, however, certainly been observed at the valves. Thus Otto[2] relates an instance in a man aged 40, and Bouillaud[3] another in a female aged 64, of superficial ulceration at the ossified semilunar valves of the aorta; and I have myself more than once found, in cases of diffuse ossification, both superficial and profound ulcers at the mitral valve, closely resembling those within arteries. Instances of *ulcers on the internal surface* of the heart's parietes are described by many authors, but unfortunately not always with due precision. They have been found almost exclusively within the left half of the heart, and, to judge from the usually very loose details, in connexion with thickening and ossification of the heart's valves, and with atheromatous degeneration of the arteries. The study of individual cases, moreover, shows that where they take a chronic course, they originate rather in a general morbid predisposition than in chronic inflammation. They occur mostly in the aged,[1] but also occasionally in young individuals,[2] and are decidedly more frequent in men than in women—circumstances which in like manner bespeak their connexion with atheromatous changes. Bouillaud looks upon perforation of the septum ventriculorum as, in many instances, the result of ulceration; and he refers to a subsequent effort at cicatrization, the smoothness of the edges in which would at first seem to militate against his assumption. Such would prove genuine examples of acquired cyanosis; little, however, as the possibility of such a process can be doubted, the instances adduced by Bouillaud (even that of Thibert) are not quite satisfactory. Nor can the joint opinion of Laennec and Bouillaud, who attribute to previous ulceration certain instances of rupture of papillary muscles, be deemed completely made out. Ulcer of the heart has been observed almost always to affect the left half; and the few authentic examples of its affecting the right side, together perhaps with those occurring in very young subjects, seem to show merely that ulceration of the inner surface may be engendered by another cause besides atheromatous disease,—such, for example, as circumscribed patches of inflammatory suppuration within the inner muscular layers of the heart. These ulcers are most frequent in the left ventricle, and occur, though very rarely, in the left auricle (in Cloquet's case, for instance). They appear to evince no predilection for any particular part of the ventricle, being found equally at the apex, the middle, and the base. In Pohl's figure the ulcerous destruction is distinctly seen to involve several papillary muscles, which circumstance is certainly in favour of the possibility before hinted at of a rupture of those muscles being caused by ulceration. They vary in size, being mostly circular and about an inch in diameter, as in Laennec's case. Sometimes, however, they are of an irregular, oblong shape, as shown in Pohl's figure. Their edges are uneven, but seldom rough and jagged, frequently simply inverted. The base is invested with a thin puriform membrane (Albers), or consists of the exposed muscular fibres, sometimes thickened by layers of fibrinous deposit. The surrounding texture is mostly softened, infiltrated with pus, or thickened with lardaceous cellular tissue, and traversed by numerous little veins, gorged with blood. These ulcers extending downwards either acquire a funnel-like shape, or by excavating the softer substance of the muscle more rapidly than they destroy the superincumbent endocardium, cause a margin of the latter to dip down into the ulcerated cavity. There is no proof of their healing, unless, with Bouillaud, we would regard certain instances of perforation of the partition wall of the ventricles, or the smooth edges of most aneurisms of the heart, as evincing partial cicatrization.

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[1] Oesterr. Jahrb. vol. xxviii, p. 65.

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[1] The changes described by Bizot appear to correspond with the state represented by Rokitansky (l. c. p. 441) as hypertrophy of the endocardium, and ascribed by him to the repeated deposition of epithelium layers.

[2] Seltene Beobachtungen (rare cases) p. 99.

[3] Maladies du Cœur, vol. ii.

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[1] Morgagni (Epist. xxvii, art. 2, 8,) in a male of 65, and in a female of 75; Cloquet (see Bouillaud) in a male of 79; Albers in a male of 73.

[2] Thomas (see Albers über den Herzriss,—on fissure of the heart,) in a boy aged 14; Broussais (see Lobstein) in a youth aged 19; Pohl. (Diss. Lips. 1808) in a man aged 45.

Progressive enlargement of an ulcer may lead either to eventual perforation of the heart's parietes, or to a protrusion of the same in the shape of an aneurismal sac. The latter case is comparatively very rare, and but few examples of it have been reported.[1] *Circumscribed aneurism of the heart* is, however, by no means to be regarded as invariably the consequence of ulcer, the previous existence of which is rarely to be inferred from the aspect of the mouth of the sac, only two instances being on record which supply unquestionable evidence of such an origin. Nevertheless, a peculiar morbid process affecting particular portions of the heart's parietes must have been at work, for we find in all the more detailed reports, that the endocardium, either extensively, or at least in the vicinity of the sac, is thickened, tendinous, semi-cartilaginous, and the like; and, in most instances, that the muscular substance has undergone cellulo-fibrous or lardaceous degeneration, and perhaps partial ossification. These changes, together with the aneurism, are ascribed by Bouillaud, Chassinat, and Thurnam, to inflammation. Breschet,[1] on the other hand, regards partial rupture of the heart as the cause of its partial dilatation; which rupture he again derives from unequable hypertrophy of the implicated cavity, with simultaneous disease of the endocardium. To this view Chassinat very properly objects that, in many, and especially in incipient cases of aneurism, no trace of rupture is discoverable; that, moreover, the hypertrophy assumed as an essential condition is either absent altogether, or at any rate often a mere consequence of the pouch-like dilatation; and that, finally, the endocardial disease is insufficient to explain either the rupture or the general dilatation. Chassinat suggests that there must necessarily be a disorganization of the muscular substance, arising from circumscribed inflammatory softening. But this superficial explanation can avail little against the fact that aneurism of the heart[2] is almost entirely restricted to the left side; (three cases alone refer to the right auricle, and even in these, it is doubtful whether it was not generally dilated;) and that in the

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[1] Not more than about 30 cases were known until the publication of Thurnam's excellent paper in the Medico-Chirurgical Transactions, vol. xxi, p. 187. Thurnam collected the cases before recorded, cited others from earlier writers which appeared to him appropriate, and added a number of new and authentic ones. He considers himself justified in admitting 58 cases of partial dilatation of the left ventricle, 11 of the left and 3 of the right auricle, and 5 of the valves of the heart. Seven others he regards as doubtful. It should, however, be remarked, that it is not fair to apply the numerical method to cases imperfectly related, or to such as have been classed under a different category by the respective authors. For undoubted cases not referred to in the above memoir, we may refer to Chassinat (Rev. Méd. 1836, p. 317); Prus (ibid. p. 344); Lombard (two cases cited by Prus); Cordet (Bullet de la Soc. Anatom. vol. ii, 1836, p. 270); Rokitansky (Oesterr. Jahrb. N. F. vol. xvii), who in his Handbook likewise refers to a whole series of instances that have come under his

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own immediate observation. Within no very long period I have myself met with three cases of aneurism of the heart, two of which presented extensive sacs at the apex of the left ventricle, the third digital impressions (as it were) on the left surface of the septum.

[1] Mém. sur l'Anevrisme faux consécutif du Cœur. 1827.

[2] Thurnam endeavours to explain the immunity of the right ventricle by the discovery of T. W. King (Guy's Hosp. Reports, vol. ii, p. 104), that the tricuspid valve closes less perfectly than the mitral, so that the blood exerts much less pressure upon the right than upon the left ventricle. But, if so, how do pouch-like dilatations occur in the left auricle, of which the entrance is free?

instance even of small ones just forming, the surrounding texture is not softened, but, on the contrary, hardened in various ways.

Partial aneurism of the heart may, as Rokitansky has satisfactorily shown, result in a two-fold manner from inflammation. Hence two varietes may be assumed. The first springs directly from inflammation of the endocardium, and of the innermost layers of muscle, and its development is accordingly very rapid. This form is by far the less common. It would appear that the softened endocardium and a portion of subjacent muscle, likewise softened and rendered turgid by inflammatory —or possibly by purulent effusion, give way, and that the blood forcibly entering the fissure, dilates it into a cavity filled with blood and detritus, which, pressing forward upon the sound portions of the heart's wall, eventually determine complete and fatal rupture. Aneurisms running this acute course never attain a large size; they affect individuals of almost every age; Rokitansky has met with them in a boy 9 years old.—The second variety is of tardy growth, although a sequel of inflammation. It is the more common of the two, and that which leads to the utmost degree of dilatation. It is dependent upon inflammation of the muscular substance of the heart, which from being converted into a fibro-lardaceous yellowish white mass, composed of dense cellular filaments,—ultimately undergoes scar-like puckering and attenuation. These changes ensuing at particular parts of the heart's parietes, and implicating either a portion only of their depth, or their whole thickness, together with both endocardium and pericardium, establish a more or less stiff, non-contractile patch; and the larger such disorganized patches, the more direct their exposure to the impulse of the blood, and the more forcible that impulse, (for instance in coarctation of the orifices of the heart,) the more readily and more rapidly will their pouch-like dilation take place. In the first form the aneurism has soft parietes and torn edges; in the second, smooth callous edges and a firm coating.

The male sex evinces a far greater proneness to aneurism of the heart than the female,—out of 47 cases, 35 were men, and 12 women. The disease is more frequent at mature age; thus, out of 42 cases, 10 referred to individuals under 30 years, and 32 to older persons. These statistics correspond upon the whole with those of atheromatous disease, which in its advanced form, as already stated, affects males in a much larger proportion than females. Partial dilatation occurs at all parts of the left ventricle, though most frequently at its apex; thus in 31 examples it occupied the apex, in 27 the base, in 15 the centre, and in 4 the septum. Aneurism is for the most part single, though occasionally more than one occurs in the same individual; thus in 4 cases there were two, in 2 three, and in one instance four.

Aneurism of the heart varies in size, from that of a cherry-stone to that of a pullet's egg;—there are, however, examples of the sac equalling or exceeding in volume the heart itself. In many cases the torn edges of the endocardium may be distinctly recognized at the neck of the tumour. Frequently, however, the opening of the sac into the ventricle is provided with a smooth, thick, tendinous, or cartilaginous, if not bony ring, which, like its whole interior is invested with a smooth membrane. This circumstanc has revived the dispute detailed under the head of arterial aneurism, touching the necessity of there being previous rupture of the internal membrane, or the possibility of simple but general distension of the parietes. We therefore refer to what was there said respecting the formation, within disorganized arteries and aneurismal sacs, of a smooth adventitious membrane, resembling the internal coating of vessels.

Upon the whole it is of no very great moment whether or not all the constituent parts of the heart's parietes and every layer of muscle contribute to form the sac; a far more important question as regards pathology is, whether a partial dilatation can take place in sound or only in diseased walls. Facts decide in favour of the latter case. Whether, in addition, the endocardium is partially sound,—whether certain layers of muscle are neither destroyed nor injured, when the dilatation commenced, is of subordinate interest. Hitherto, too much attention has been paid to these distinctions of form, and too little to other circumstances calculated to elucidate the fundamental disease. It may be observed, incidentally, that Thurnam has demonstrated the occurrence in the heart of most of the forms of aneurism that affect the arteries; although in some instances in rather a forced manner.

The saccular dilatations[1] either bulge over the external surface of the heart, or lie imbedded, as it were, within its walls. In the latter instance they are very small, open with a wide orifice into the ventricle, and are bounded by all the layers of the cardiac parietes. Thurnam, however, describes in his third case a non-projecting aneurism of this kind, which at its convexity was bounded only by the thickened endocardium, and the serous investment of the heart. The large and prominent tumours exhibit a narrow and as it were constricted neck,—rarely traces of muscular substance throughout their whole extent—this being attenuated at the very mouth, and gradually decreasing, until at length the principal remaining part of the tumour is merely encased within the thickened serous investment of the heart. In isolated cases a steatomatous, cartilaginous, and osseous degeneration of the parietes of the sac has been observed. In proportion as the aneurism makes way externally, it ere long coheres with the pericardium, and if the pressure lasts, amid cellular degeneration of the serous membranes, and lardaceous thickening of the cellular texture, enters into further adhesions with the lung, and even with the parietes of the thorax (Harrison's case). Sometimes partial dilatations occur in the vicinity of the aorta, and in connexion with it (Hodgson, Hope, and Lombard's cases). In one instance (Lombard) an aneurism of this kind had, through perforation, established an anormal communication between the left auricle and ventricle; in another (Rokitansky), an aneurismal sac occupying the septum communicated with the right auricle.—As in arterial aneurism, so in cardiac sacs, fibrinous coagula become deposited in layers, from stagnation of the blood, whereof the external strata are the firmest, and adhere to the parietes of the tumour, the remainder becoming softer the more inward they are, until finally they are coated with clotted blood. This fibrinous deposit is thick in proportion to the size of the sac and to the narrowness of its mouth; and in small aneurisms having a very ample communication with the ventricle, is generally entirely wanting.[2] Curative efforts of nature have not been observed in these partial dilatations. They mostly prove fatal amid the usual symptoms of heart disease, and commonly through bursting of the sac.

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[1] For figures, see Walther, Breschet, Carswell (l. c.) and Cruveilhier (livr. xxi, vol. iv; xxii, vol. iii, f. 2.)

[2] Thus in Carswell, fasc. ix, pl. ii, fig. 2, 3.

But one instance of *aneurism of an auricle* is known, and that of the left (Chassaignac's case.) Other cases refer to the diffuse form,[1] and accord more or less with general dilatation. They arise from coarctation of the auriculo-ventricular orifice. Finally, we have to notice aneurism of the heart's valves, two instances of which (Morand, Laennec,)[2] are upon record as affecting the mitral valve. Thurnam describes three other examples,—one of the mitral valve, another of the valves of the aorta (of which, owing to an original fault of development, there were but two,) and a third of the tricuspid valve in a cyanotic subject, there being a communication between the two ventricles, at the upper part of the septum. The saccular dilatations of the mitral valve pointed towards the auricle,—those of the semilunar valves towards the ventricle. In three cases of valvular aneurism there were perforations; in all there were thickening, fibrinous, warty deposits, and calcareous incrustations, either at the valves, or elsewhere.

According to Rokitansky, valvular aneurism always proceeds from inflammation, and is brought about either by the immediate rupture of one of the lamellæ of the valve, and pouch-like distension of the other through the ingress of blood, or, from pus forming between the lamellæ, or insinuating itself thither from the walls of the heart, and causing rupture of the lamella directly exposed to the impulse of the circulation. The saccular distension is in most instances inconsiderable, although it has been known to attain the size of a walnut. (Thurnam.)

These partial aneurisms are associated with a variety of changes in the heart itself. In a vast number of cases there are simultaneous ossifications, implicating the valves, and extending more or less into the aorta. The endocardium is invariably thickened, of a yellowish-white aspect, and a tendinous or semi-cartilaginous character. The papillary muscles are attenuated and atrophied, which is also here and there the case with the substance of the heart's walls; or the latter are found partially converted into a yellowish-white, or a darker nearly homogeneous mass, resembling boiled white of egg. In certain instances there is at the same time ulcer of the heart. (Bordet, Lombard.) Hypertrophy and dilatation have been very frequently observed to affect the remainder of the heart's substance, either generally or partially; in the majority of cases the left ventricle alone; in three only of the examples reported is it expressly stated that the heart was of natural dimensions. The hypertrophy was however seldom very considerable, and not always uniform, inasmuch as certain patches were here and there palpably attenuated. Here it is to be borne in mind that the column of blood must have exercised unequal pressure on different portions of the heart's parietes,—these having suffered various—in one place albuminous, in another, fibro-cellular—degeneration.

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[1] That of Elliotson, for instance.

[2] Carmichael also relates an instance of aneurism of the aortal valves. (Dublin Journ. July 1841.)

When inflammation or atheromatous disease induce *rupture of the heart,* more especially if it be through the medium of abscess or ulcer, the organ is mostly found to be of natural dimensions, though sometimes dilated, with simultaneous attenuation, or even hypertrophy of its walls. Its substance is lax, and softer, or denser, but at the same time more friable than usual, just as in endocarditis, or in fatty infiltration of the heart. This prevails either throughout, or at certain portions; the immediate vicinity of the fissure is usually the seat of various morbid changes. In fifty-four cases of spontaneous rupture of the heart, collected by Pigeaux,[1] there was found, seven times, dilatation of the implicated cavities, with equable attenuation of their parietes; thrice partial attenuation, and thirteen times general softening thereof; and, finally, twenty-seven times organic mischief of various kinds in the neighbourhood of the rupture. Hence it appears that neither Rostan, who regarded rupture of the heart as altogether spontaneous and independent of any previous pathological change of texture, nor Bland, who affirmed gelatinous softening to be the exclusive cause, have been right. In thirty-six cases the long diameter of the rupture was parallel with the main fibres of the heart's muscle; and these fibres were transversely ruptured in thirteen cases only. Pigeaux concludes from this, that the rupture is of a passive nature, and occurs *during diastole.* This seems the more likely, when we reflect that during systole the valves are open and the egress of blood facilitated, whilst during diastole the valves are closed, the walls of the cavities dilated, and the muscular fibres upon the stretch. It is thus explained why precisely the thickest portions of the walls, and the left ventricle, are most prone to rupture, as forty-five examples actually show to be the case. The valves, tendinous cords, and sometimes the papillary muscles are, as already stated in the section Endocarditis, likewise subject to laceration.

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[1] Journ. Hebdomadaire, vol. viii, No. 104, 1832, (Nouv. recherches sur l'étiologie des ruptures spont.,) &c.

When the rupture, instead of occurring suddenly, ensues by degrees from within outwards, there is considerable bloody effusion between the muscular fibres of the heart's walls, constituting positive *apoplexy of the heart;* this is not, however, as Cruveilhier contends, the cause, but rather the consequence of the rupture. It often happens that the perforation, though single on the inside, opens with several orifices externally, the heart's wall having given way in a very oblique or even in a zigzag direction.[1]

In connecting rupture of the heart with inflammatory or atheromatous degeneration, we must not omit to mention that all cases cannot be assigned to this twofold source, not a few being obviously referrible to fatty degeneration, to the various forms of softening, or finally to simple dilatation and attenuation of the heart's walls.

While most cases of this accident—not of traumatic origin—are, according to Laennec, the result of ulceration, it might be asserted, with no less accuracy, that most cases of ulcer of the heart *end* in perforation. All the above cited were examples of this, and in many other instances of rupture that have been reported, ulcer must certainly have existed although overlooked.

The anatomical details contained in dissertations upon rupture of the heart distinctly indicate its frequent connexion with atheromatous disease; yet it should be observed that the atheromatous changes, by determining impediments to the circulation at the orifices of the heart, are to be regarded less as direct than as occasional causes of the rupture. Thus Albers[2] says: "The valves not rarely contained bony masses; and deposits of the same material were found within the large arteries in the shape of osseous discs. Dilatation of the cavities of the heart, and thickening of the walls, are results neither constant nor to be relied upon, having been in general either undiscovered or but imperfectly described." Albers believes in the possibility of rupture of the heart without previous ulceration, and that simply as a consequence of partial ossification. He says (p. 207): "The deposition of bony matter is very frequent at the upper margin of the left ventricle, and in the vicinity of the mitral valve, in which situation the occurrence of fissure is the most common." Again, p. 208 (after Portal): "Without doubt the ossifications formed at the mouth of the great artery of the left side of the heart, were the source of the rupture." Pigeaux is likewise of opinion that atheromatous, steatomatous, tubercular, and cancerous degeneration are its most frequent sources.

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[1] Cruveilhier, livr. xxx, pl. iv.

[2] Horn's Archiv, 1832, vol. i, p. 201.

From the cases collected by himself, Albers infers rupture of the heart to be a disease of advanced age, most of those cases relating to persons sixty or seventy years old. Twenty-two of them affected the left, three the right, and three both chambers. According to Pigeaux, forty-four cases affected the left, eight the right ventricle; one the left, and one the right auricle.

## SECT. V.—VEGETATIONS UPON THE VALVES, AND UPON THE INNER SURFACE OF THE HEART GENERALLY.

Laennec divided these growths into "*warty*" and "*spherical.*" The latter are often congenerous with the granulations described under Endocarditis, and mostly result from local inflammation, or at least from some more or less remote inflammatory process, whose product, conveyed by the blood, forms an organic union with the tissue upon which it becomes deposited. They are soft, spherical or oval bodies, from the size of a pea to that of a pigeon's egg,[1] with simple or locular cavities containing every gradation from mere coagulated blood to serum. They adhere to the inner surface of the heart by a pedicle, attached either by numerous roots to the fleshy columns, or by a broad adherent base to the endocardium. Sometimes the granulations accumulate at the valves into soft, mulberry-like, solid masses, loosely implanted on the endocardium, with which they have no vascular connexion at first, although they may acquire it by and by. (Lobstein's *fausses excroissances verruqueuses.*)

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[1] See for example, Carswell, fasc. xi, vol. ii. fig. 1.

The various origin and forms of fibrinous coagula within the heart having been already pointed out, the occasional transformation of those engendered during life, into the aforesaid spherical growths, independently or not of endocarditis, deserves notice. Their development is generally coincident with that gradual diminution of the heart's action which precedes death. The coagula enlarge in a stratiform manner, and display on microscopic examination an amorphous granular structure. It is not improbable that when developed during a *brief interval* of diminished heart's action, and when comparatively very minute, they may afterwards gradually liquefy in the circulating fluid, and so disappear. On examining coagula again of longer standing, we find the granular congeries of fibrinous molecules disposed in tolerably regular parallel fibres, somewhat stouter than those of cellular tissue, but less sharply defined, and not so undulating in shape. If any local inflammation contributes to the development of the coagula, we mostly observe granule-cells mingling with the amorphous fibrinous masses. It appears, however, that to be convertible into the spherical growths, the coagula must necessarily be of small dimensions, the inflammation which gave rise to them slight and transient, and the opportunity for their fastening somewhere upon the heart's parietes immediate. They do not, indeed, even then, enter into any vascular connexion with the latter, but may nevertheless, when in direct contact with the plasma of the blood, undergo further grades of development. Their external superficies first becomes rounded off by partial solution, they shrivel up into a very small compass, are rendered cellular through inward softening, and contain, in the first instance, dissolved fibrin, or serum. Subsequently their cavities fill up, the whole acquires greater solidity, and the aspect of a vegetation firmly seated upon the relaxed endocardium. Ultimately they may become converted into a calcareous mass. Concrements of the kind are sometimes found attached more especially to the auricular valves; they are moveable and brittle, mostly isolated, unaccompanied by degenerations of other kinds, and seldom larger than small peas. The several grades of development of the coagulum cannot indeed be traced in uninterrupted connexion, but the above description of their progressive formation is authorized by a comparison of cases.

Amongst the forms may be recognized certain soft *spherical* whitish *coagula*, either interwoven by means of simple or multiple pedicles amongst the fleshy columns, or else agglutinated as it were, by a broad base, to the endocardium. They are mostly hollow, contain puriform fluid, and vary in size from that of a pin's head to a hazel nut. In several cases which came under my notice I was unable to detect any blood-vessels, although the limited patches of the endocardium to which they adhered were rough and reddened (see Endocarditis). J. F. Meckel who, in his 'Tabulæ Anatomico-Pathologicæ' (Fasc. i, pl. 7,) represents one of these cases, regards them as true polypi of the heart, and their puriform contents as a product of inflammation of their substance. We should be extremely cautious in opposing so great an authority, but equally scrupulous in testing facts, and as I have been unable to detect any vascular link between the endocardium and the parietes of these spherical masses, any more than F. Nasse in two similar cases (see Leichenöffnungen, pp. 16, 24), I should view them, if not as products of endocarditis, at any rate as a sequence of the absorption of pus, or of the matter of softened tubercle into the blood. The latter explanation is perfectly applicable to the cases observed by Nasse and myself, in all of which the lungs contained more or less of softened tubercle. (See Phlebitis.)

Indeed the fluid contents of these hollow spherical coagula, though frequently presenting at first sight the greatest resemblance to pus, appear perfectly distinct from it when examined with the microscope; for there are no pus-corpuscles, but merely an amorphous, finely granular mass, consisting of dissolved fibrin, as stated by Gulliver. In many instances the fluid is of a reddish or a drab colour, according to the number of blood-corpuscles entering into its composition. I have met with these spherical coagula more frequently in the left than in the right ventricle,—in one instance in both together,—always at the apex.

The proper *warty vegetations* of Laennec are very like syphilitic condylomata, for which they were mistaken until Kreysig cleared the matter up. They occur only at the valves, sometimes as little acuminated cones, arranged in rows or in groups; sometimes as warty, or cauliflower-like, soft masses; sometimes, again, as cellular, cockscomb-like growths, covered with nodules and filaments.[1] It is impossible to determine whether they are invested with endocardium; their attachment is however tolerably firm, and in some cases decidedly vascular.

Finally, we would advert to a change observed in the free margin of the auricular valves, not, strictly speaking, a vegetative growth, or a pathological product, since it is present in at least one half the number of dead bodies that are examined. We allude to the little fibrous or fibro-cartilaginous bodies, not bigger than a lentil, occurring more particularly at the point of insertion of the tendinous cords, and resulting apparently from simple hypertropy of the fibrous texture, so abundant in the mitral and tricuspid valves. These indurations form all along the margin of the valve, and never ossify.

The faulty states of the valves just described, have no connexion whatever with atheromatous disease, although Albers and others attest their coincident occurrence. It would appear that neither sex nor age dispose to their development.

## SECT. VI.—ATROPHY OR INADEQUACY OF THE HEART'S VALVES.[2]

This morbid condition was not properly understood until the publication of Kingston's paper in the 'Medico-chiurgical Transactions' (vol. xx, p. 90), although alluded to by several of the earlier authors on disease of the heart, and clearly exemplified in certain recorded cases.[1] The anatomy thereof as affecting the semilunar valves, has been amply illustrated by Bizot (l. c.)

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[1] See figures in Carswell, fasc. xi, pl. ii, fig. 3; Albers, section vi, vol. ix, fig. 1; Froriep, Klin. Tafeln (after Elliotson) pl. xxix.

[2] By inadequacy of the valves is practically meant all those conditions upon which depend imperfect closure of the heart's orifices, and, consequently, regurgitation from the ventricles into the auricles,—from the great arterial trunks into the ventricles. In this sense, ossification, rigidity, corrugation, inversion, and rupture of the valves or of their bonds of attachment (the papillary muscles and tendinous cords of the mitral and tricuspid valves) as well as adhesions and ulceration of the valves may give rise to their inadequacy. The disappearance of the pouch-like sinuses at the base of the auricular valves, from adhesion, &c., to which Skoda first directed attention, belongs more particularly to this category. A relative insufficiency will, as observed by Hope, result if, whilst the ventricles and the venous orifices are preternaturally distended, the auricular valves retain their natural proportions, so as to be

That so little regard was formerly paid to this atrophy or insufficiency of the valves, may have been owing, partly as Kingston remarks, to its easily escaping observation, as it requires for its detection attentive research,—partly to the previous want of a groundwork for an exact estimate of form and dimensions, such as is now afforded by coupling physical research with anatomical data.

Of this insufficient state of the valves, two forms have been recognized; namely, a shortening, and a net-like condition; the former in the mitral and tricuspid, the latter in all the valves of the heart. Neither of the forms being rare, it is to be hoped that their influence upon the general health, and upon other morbid states of the heart, will, ere long, be made the subject of distinct investigation. Since my attention has been directed to them by Kingston's paper, I have repeatedly met with examples, and am inclined to believe that their importance in a pathological point of view is not uniformly great.

*Shortening of the auricular valves* may or may not be associated with attenuation. It is sometimes very considerable; Kingston found the posterior fold of the mitral valve reduced to 3, nay to 1½ lines in length, (the natural dimension being 9 lines,) and certain portions of the tricuspid valve (from 8 to 11 lines long in the normal state) only 3 lines long. When all the folds of a valve are shortened (of which I have seen but one example, on the right side of the heart), the systole must be necessarily accompanied by regurgitation of blood into the auricle, and the latter be consequently dilated,—the ventricle both dilated and hypertrophied. If, on the other hand, a single fold alone is shortened, the posterior one of the mitral valve for instance, the anterior will still suffice in most instances for

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no longer capable of effecting complete closure. The organic defects treated of in the present Chapter are, however, unconnected with the secondary diseases just referred to, and must therefore be described independently. They constitute true atrophy of the valves.

[1] For instance, in Nasse's Leichenöffnungen (Examinations of the dead body) page 106.

closing the auricular orifice; this will be impossible, however, if the fold nearest to the mouth of the artery be curtailed. Thus, in the afore-cited case of Nasse, the right auricle was found much dilated, and its parietes attenuated,—the ventricle dilated and hypertrophied; two folds of the tricuspid valve were much shortened; while of the third, nearest to the septum, nothing remained, except a small floating lamella. The auricular orifice was at the same time unusually capacious.

The cribriform or net-like condition is rare in the auricular valves. Kingston saw four instances in which the number and extent of the meshes must have necessarily caused some disturbance in the circulation. Perhaps we might refer hither a case mentioned by Laennec[1] as aneurism of the mitral valve,—the long fold of which presented an aperture 4 lines in width, with its margin projecting half an inch into the auricle. The *arterial valves* very frequently exhibit net-like perforation. Out of 157 subjects examined by Bizot, the valves of both arteries were thus impaired in 36 cases, and in other 36 the valves of either the one or the other artery almost in equal proportions.[2] Before the sixteenth year this condition is comparatively rare; it is most common from that period to the thirty-ninth year, and of mean frequency during the remainder of life. The perforations are in general immediately subjacent to the free margin of a semilunar valve, and vary in size from that of a pin hole, to that of the entire space between the sesamoid tubercle and the lateral attachment of the valve. The net-like alteration does not extend to the inferior two thirds of the membranous portion of a semi-lunar valve, except in rare instances; and then the valve is altogether disqualified for use. If, on the contrary, this be limited to the immediate neighbourhood of the upper margin, no disturbance of the circulation can ensue; for, on closure of the mouth of the artery, the margins of the three semilunar valves overlap one another so amply as to prevent any mischief arising from a few perforations. It is self-evident why, under these circumstances, symptoms of heart disease have only in rare instances been observed; indeed, in the great majority of cases, there has been no functional disturbance whatever. (See Bizot.) Even, however, when such symptoms have been discovered, they cannot uniformly be ascribed to the atrophic state in question; for in Kingston's cases, it appears that there was concomitant organic disease of the heart of another kind. According to my own experience, the net-like condition of the arterial valves (though not the shortening of the aricular valves) rarely occurs in conjunction with advanced atheromatous disease. The two affections do not, however, necessarily exclude each other; as is shown by one of Kingston's cases, in which the aortic valves partially coalesced with each other, and were for the most part thickened, but presented several large perforations with surrounding attenuation.

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[1] On Mediate Auscultation. See transl. by Dr. J. Forbes, 4th edition.

[2] I have met with this condition less frequently than Bizot, and refrain from making any numerical statement, because, not attaching to it the same pathological importance, I have not sought for it perhaps with like assiduity. In order to find it, it is merely requisite to raise up each valve separately with the handle of the scalpel.

Insufficiency of the valves cannot be generally reckoned a congenital malformation, since, according to Bizot, it is far less frequent in children than in adults. We must view it, with Kingston, as an acquired atrophy, gradually engendered, under favorable circumstances, by the perpetual impulse of the column of blood against the valves. Here, as in other organs, continued pressure is productive, now of increased absorption and of wasting, now of augmented secretion and thickening. At first the membranous portion of the valve wastes,—the fibrous texture distributed throughout in individual fibres, remaining intact to constitute the cords of the meshes. This explanation suffices for cases in which the perforations occur at the centre of the valve's parietes, not for those in which the net-like condition is found near the free margin of the semilunar valves, which, fully overlapping each other, are little, if at all, subject to pressure. It is perhaps precisely because this part of the valve is no longer engaged in the true valvular function, that it wastes thus; just as the eustachian valve is found in adults perforated in a net-like fashion. The etiology of the above conditions is not yet thoroughly made out, and ample room is still left for conjecture and theory.

### SECT. VII.—DISEASED CONDITION OF THE HEART'S CAVITIES, WITH REFERENCE TO THE DEPTH OF THEIR WALLS,—(HYPERTROPHY AND ATROPHY,) AND TO THEIR CAPACITY,—(DILATATION AND COARCTATION.)

These, mostly secondary, though sometimes primary affections, have ever attracted the attention of pathologists, on account of the serious symptoms to which they give rise. They have received different appellations, corresponding to the numerous varieties which have successively been recognized during life and after death. The principal forms are comprehended in the following schedule:

1. *Dilatation of the heart's cavities, with thickening of their walls.*

Dilatation with hypertrophy. Eccentrical hypertrophy. Active aneurism of the heart. Incomparably the most frequent form.

2. *Dilatation of the heart's cavities with natural thickness of their walls.*

Simple aneurism of the heart. Simple dilatation. In like manner to be regarded as dilatation with hypertrophy, and as active aneurism of the heart, since dilatation of the cavities necessarily implies general increase of muscular substance. Occurs rarely in this simple form

3. *Dilatation of the heart's cavities with attenuation of their walls.*

Dilatation. Passive aneurism of the heart. Rather frequent. Affects the right division of the heart more particularly.

4. *Thickening of the walls without dilatation of the cavities.*

Simple hypertrophy. Rare,—perhaps still problematical.

5. *Thickening of the walls with coarctation of the cavities.*

Concentrical hypertrophy. Partial only,—doubtful according to some pathologists.

6. *Attenuation of the walls without dilatation of the cavities.*

Very frequent.[1]

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[1] The late Dr. Hope, in his work on Diseases of the Heart, gives the following summary of these forms of disease. *Ventricles:* 1. Hypertrophy and dilatation of the

Most of the above forms may be general, involving all the cavities of the heart,—or partial,—limited to one or more. In the latter case there may exist the most varied combinations of forms, to specify which would serve no useful purpose. It is to be observed, in a general way, that the auricle and ventricle of the same side are in most instances affected in common; and that the left division of the heart is more disposed to hypertrophy,—the right to dilatation.

In order to estimate with accuracy the relative proportions of the heart in disease, it is indispensable to be familiar with its dimensions in health. In this respect pathological long suffered from the neglect of physiological anatomy,[1] measurement being for the most part restricted to a rude comparison with the fist of the dissector, and the dimensions of parts like the heart's orifices, simply determined by the eye, or committed to memory. Lobstein and Bouillaud made the first attempt to reduce to a fixed physiological standard the weight and dimensions of the entire heart and of its several parts; though, as regards the ventricles and their orifices, these relations were first discriminated by Bizot (Mém. de la Soc. Méd. d'Observat. vol. i, p. 262.)

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left ventricle, together with a slighter degree in the right ventricle. 2. Hypertrophy and dilatation of one, chiefly the left, ventricle, with simple dilatation of the other. 3. Simple dilatation of both ventricles. 4. Simple hypertrophy of the left, together with hypertrophy and dilatation of the left ventricle. 5. Dilatation and attenuation of the left ventricle. 6. Hypertrophy and coarctation of the left ventricle. 7. Hypertrophy and coarctation of the right ventricle. *Auricles:* 1. Dilatation, especially of the right auricle, from accumulation of blood. 2. Dilatation and hypertrophy. 3. Hypertrophy, with coarctation.

[1] I began, several years ago, to pay attention to the width of the pulmonary artery relatively to that of the aorta. In E. H. Weber's edition of Hildebrandt's Anatomy, and in J. Müller's Physiology, it is asserted that the former is narrower by one third than the latter, and from this statement many physiological inferences have been drawn. Great was my astonishment, however, to find that, on the contrary, the pulmonary artery is somewhat more capacious than the aorta; yet, on repeating the trial in a multiplicity of cases, I invariably arrived at the same results. Whilst I was thus engaged, Bizot's comprehensive Researches were published, and proved far in advance of my own. Nevertheless, whenever I found time, I continued my admeasurements upon a somewhat more comprehensive principle and improved method. My results, based upon 122 admeasurements, almost entirely coincide with Bizot's, and are richly suggestive in a pathological point of view. It should be stated that the measurements from which the general results are taken were made on subjects who, during life, had betrayed no symptoms of diseased heart.

Bizot found that from birth to extreme age the volume of the heart is continually on the increase, this being obvious up to the 29th year, and subsequently, though not immediately perceptible to the eye, still appreciable by actual measurement. This growth of the heart mainly depends upon the progressive enlargement of its orifices, and the increased thickness of the walls of the ventricles. The gradual thickening is most apparent in the walls of the left ventricle,—less so in those of the right, where it is sometimes not at all perceptible. Thus in the female subjects examined by myself, the average thickness of the walls of the right ventricle between the thirtieth and thirty-ninth year, was $1\frac{3}{5}$ of a Parisian line; between the sixtieth and sixty-ninth year, $1\frac{2}{3}$; and between the seventieth and seventy-ninth, $1\frac{1}{2}$. The progressive enlargement of the two auriculo-ventricular orifices is tolerably uniform; that of the two arterial mouths differs; both increase equally until the meridian of life, but the aortic orifice enlarges more rapidly in advanced age than that of the pulmonary artery, so that in old persons the latter is even narrower than the aorta. This variation I have met with earlier in males than in females; (in the former from the 50th year upwards, in the latter not till after the sixtieth; to a still later period of life it no longer applies. In children the orifices of the two arteries continue equally capacious until the sixth or even until the tenth year. The right half of the heart differs from the left in the greater capacity of its cavities, and the greater width of its walls. The difference of sex is striking: first, all the dimensions are smaller in women than in men; secondly, the venous orifices are relatively smaller, but the mouth of the pulmonary artery proportionately of greater width. Again, it is certain that in very tall men and women the heart is comparatively smaller than in those of shorter stature; and larger in broad than in narrow shouldered persons.

I shall in this place briefly enumerate the average proportions of the volume of the heart in adults, and refer the reader for further particulars to Bizot's Essay, and to a memoir which I am myself about to publish.

In subjects between the thirtieth and forty-ninth year, the heart presents (according to Bizot):

| | Parisian Lines. In men | Parisian Lines. In women |
|---|---|---|
| A length of | $43\frac{3}{23}$ | $41\frac{2}{27}$ |
| Breadth | $47\frac{18}{23}$ | $44\frac{1}{27}$ |
| Depth | $17\frac{4}{23}$ | $14\frac{4}{27}$ |
| Length of left ventricle | $29\frac{11}{23}$ | $31\frac{16}{17}$ |
| Breadth of ditto | $53\frac{4}{23}$ | $46\frac{4}{17}$ |
| Length of right ventricle | $37\frac{13}{23}$ | $33\frac{13}{27}$ |
| Breadth of ditto | $83\frac{10}{23}$ | $76\frac{17}{27}$ |
| Thickness of the walls of the left ventricle at the base | $4\frac{17}{46}$ | $4\frac{1}{9}$ |
| Ditto at the middle | $5\frac{1}{11}$ | $4\frac{27}{54}$ |
| Ditto near the apex | $3\frac{13}{23}$ | $3\frac{6}{27}$ |
| Thickness of the septum of the ventricles at the middle | $4\frac{21}{23}$ | $4\frac{11}{27}$ |
| Thickness of the walls of the right ventricle at the base | $1\frac{39}{46}$ | $1\frac{19}{27}$ |
| Ditto at the middle | $1\frac{7}{23}$ | $1\frac{13}{54}$ |
| Ditto near the apex | $\frac{45}{46}$ | $\frac{25}{27}$ |
| Width of the left auriculo-ventricular orifice | $48\frac{9}{22}$ | $40\frac{17}{26}$ |
| Ditto of the right | $54\frac{5}{23}$ | $47\frac{4}{27}$ |
| Width of the origin of the aorta (above the valves) | $30\frac{20}{23}$ | $28\frac{3}{27}$ |
| Width of origin of pulmonary artery | $31\frac{12}{23}$ | $29\frac{1}{3}$ |

Hence it follows that, contrary to the opinion of most pathologists, incipient hypertrophy is present, wherever the thickness of the walls of the left ventricle exceeds in men 6, in women 5 Parisian lines; that of the walls of the right ventricle, in men 3, in women $2\frac{1}{2}$.[1] It is to be regretted that equally exact measurements of the auricles are still wanting. Bouillaud communicates four instances of healthy males, in which he measured the auricles. In one, aged sixteen, he found the walls of the left auricle 2 lines thick; in another aged thirty-four, $1\frac{1}{2}$; in another aged sixteen, $1$—$1\frac{1}{2}$; in the last, aged twenty-five, $\frac{3}{4}$ to 1 line: those of the right auricle respectively, $1\frac{1}{2}$, 1, $\frac{1}{2}$ lines. These proportions are evidently too high; either the hearts must have been diseased, or the fleshy columns comprehended in the measurement.

In true hypertrophy, the muscular substance of the heart augments to an extraordinary degree; the walls of the left ventricle sometimes attaining a thickness of 14, those of the right of 7 lines, and upwards. This increase may affect the muscular layers collectively, or limit itself either to the external ones, which is most usual, or else to the fleshy bundles and the papillary muscles. The morbid process of nutrition may implicate all parts of the heart equally, or one auricle or ventricle in a preeminent degree; nay, examples occur of isolated portions only of a ventricle being hypertrophied. It may be asked whether the process be one of *mere* nutrition?—in other words, whether there be an increase of *healthy* muscular fibre, or whether the substance deviate qualitatively as well as quantitatively from the natural standard. As yet, this point has not been decided, either by chemical or microscopic research. Albers (Erlæuterungen zu seinem Atlas, iii, p. 59,) conceives that hypertrophied hearts, after long maceration in alcohol, are more easily torn than sound hearts, similarly treated; but upon this general assertion little reliance can be placed. More value would attach to the observation, if confirmed, that the individual fibres separate less readily from each other in hypertrophied, than in healthy hearts. In many cases the hypertrophied substance exhibits the same colour and consistency as the natural, although for the most part the consistency is augmented; the muscular texture denser and firmer, particularly in the right ventricle, and at the same time of a deep red, approaching to a violet colour; (so figured by Carswell, fasc. ix, pl. ii;) I have been unable to detect, by the microscope, anything preternatural in the character and disposition of the muscular fibres. In other instances the muscular substance has lost its peculiar softness and pliability, becoming compact or unyielding, brittle or easily torn, whilst the whole mass, or at least certain patches assume a dingy brownish, or dingy gray, or even a grayish yellow hue, which, at a first glance, would seem to result from matter effused between the muscular fibres. Again, the colour of the heart is of a faint dingy brown, approaching to yellow, and its consistency vastly diminished, when death has shortly been preceded by an inflammatory state. The absolute weight of the heart is necessarily increased in hypertrophy; it may amount, according to Lobstein, to upwards of two pounds; (vol. ii, p. 419;) whereas, according to the same authority, a healthy heart weighs only from 8½ to 10 ounces.

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[1] The fleshy columns are, of course, not included in these dimensions.

The cavities of a hypertrophied heart are for the most part also dilated. Where the hypertrophy is general, and at the same time intense in degree; this is invariably—where it is partial, most commonly—the case, so that Bouillaud reckons for every twenty cases of eccentrical, but one of simple hypertrophy, and that one affecting the left ventricle rather than the right. The mutual relations of the different cardiac cavities are almost wholly dependent upon the occasional causes of hypertrophy or dilatation. The malady seldom develops itself in all the cavities at once, but mostly originates in one, and then gradually proceeds, in conformity with mechanical laws, to the others. If it commence on the right side of the heart, it need not necessarily implicate the left, where any existing hypertrophy may be due to some other cause. When, however, it begins on the left side, a subsequent affection of the right is almost always the result, more particularly where it is of mechanical origin.

*Concentrical hypertrophy*, first described by Bertin, is not universally admitted. Cruveilhier always found it in individuals who had died suddenly, for example by the guillotine, and he believes it to be produced by the contractility acting with full vigour up to the moment of death. He was able, by artificial dilatation or by maceration, to restore hearts thus thickened, to their natural condition. In the twenty-first volume of the 'Medico-Chirurgical Transactions,' Budd disputes, likewise from experiment, the possibility of concentric hypertrophy establishing itself during life, and refers its occurrence after death to anæmia, the effect of which would be to contract the walls of the heart more closely; wherefore its frequency in persons that have been beheaded, or destroyed by cholera. The affection is produced by malformation, where a communication exists between both sides of the heart, giving rise to inordinate activity in either. The less active then exhibits thickening of its muscular walls, coarctation of its cavity, and also of its arterial orifice, with whose capacity that of the cavity must needs correspond; indeed hypertrophy with coarctation of the implicated cavity is physically impossible, so long as the outlet retains its natural dimensions. From cases, however, which have come under my own eye, the question does not appear to be yet satisfactorily settled, particularly as relates to coarctation of the ventricles from enlargement of the fleshy columns and papillary muscles.

Simple *dilatation* without hypertrophy, and dilatation with attenuated walls, sometimes occur in a slight, rarely in a considerable degree. The former (*simple aneurism of the heart*) is always associated with dilatation of the arterial orifices, and very frequently with preternatural width of the whole course of the aorta and of the pulmonary arteries. The second form (*passive aneurism of the heart*) is concurrent with enlargement of the auriculo-ventricular orifices, and of the veins opening into the heart. The right division of the heart presents this condition more frequently than the left. Comparatively speaking the auricles dilate most, the right commonly in consequence of general, but more especially of pulmonary stagnation of the blood. The left, owing to coarctation of its passage into the ventricle; both, owing to insufficiency of the tricuspid and mitral valves. The muscular substance of the heart is lax and soft in such cases, sometimes dark coloured, at other times pale, and loaded with fat. The fleshy columns are attenuated, elongated, and parted as it were; the septum is least of all changed. Simple idiopathic dilatation ought not to be confounded with a condition of the heart brought on by diseases which are attended with great decomposition of the blood, such as malignant typhus, phlebitis with absorption of pus into the blood, scurvy, &c. Here the heart is pale and flabby, its cavities enlarged from relaxation, its coronary veins either gorged with blood, or empty, their track being marked by imbibition.

In every instance of dilatation, with and without hypertrophy, the *shape* of the heart is *altered.* The apex disappears, being rounded off. When the ventricles are alone affected, the heart acquires a somewhat globular form; it is more cylindrical, though still rounded inferiorly, like a leathern purse, when all the cavities, or both of one side only are implicated. In this latter case the dilated ventricle becomes lengthened at its lower part, and generally alone forms the heart's apex, whilst the septum between the ventricles projects archwise into the dilated, but not hypertrophied ventricle. The spherical shape is the more distinct, the more the dilatation or hypertrophy of one side predominates, except in concentric hypertrophy; it is less marked in simple aneurism of the heart, and scarcely appreciable in simple, uniform hypertrophy of the totality of the heart. With respect to the shape of the ventricles individually, the left suffers the least change, appearing more pouch-like where dilatation, more stomach-shaped where simple hypertrophy prevails. In the right ventricle, the increase of volume is most palpable in the portion facing the pulmonary artery, it is therefore most convex at its anterior surface and at its base. In dilatation with hypertrophy of the left ventricle, the dilatation is in like manner confined to the anterior portion, the cavity being at the same time obtruded upon by forcible bulging forward of the septum.

Where the hypertrophy is considerable, the heart is, for the most part, *obliquely* placed on the left side of the thorax, with its apex directed to the left, and its base to the right. Hereby, the vessels entering and leaving the heart are necessarily diverted from their proper course; a circumstance fraught, in all likelihood, with no little disturbance to the circulation. When the hypertrophy attains a high degree, unless something happens to prevent or modify the displacement, the heart partly sinks towards the epigastrium, pressing down the diaphragm, partly falls back towards the vertebral column, so as to embarrass the lung.

In hearts much dilated, and especially if hypertrophied at the same time, we find after death the cavities loaded with a quantity of thick clots of black grumous or pultaceous blood. With these clotted masses the fibrinous coagula have no connexion whatever, the latter being present in quantity proportionate to morbid processes of a different kind. The auricular sinuses are sometimes filled with stratiform fibrinous coagula.

A variety of opinion is entertained touching the circumstances which determine the development of the diseases above described. Lobstein's explanation is perhaps the clearest, and to it we shall frequently revert. Bouillaud, who derives almost all cardiac diseases from inflammation, refers hypertrophy to the same source, more particularly to endocarditis, as defined by himself. Thus far he agrees somewhat with those pathologists who maintain hypertrophy and dilatation to be of development purely mechanical, and dependent, for the most part, upon thickening, adhesions, ossifications of the cardiac valves and consequent coarctation or else widening of the orifices; conditions regarded by Bouillaud as emanative from endocarditis.

Most cases, especially of *partial* hypertrophy or dilatation, depend unquestionably upon faults of the heart's orifices, although these vary greatly as to origin and character. Thus ossifications of the valves exercise great influence, not only because they diminish the orifice and thus impede the passage of the blood, but because they disqualify the valves for effecting perfect closure of the passage, so as to prevent regurgitation. The consequence is dilatation with hypertrophy of the left auricle, where the mitral valve is thus ossified, and the same condition of the left ventricle and auricle at the same time, where the semilunar valves of the aorta are at fault. It follows from what has been already stated respecting atheromatous disease, that these conditions are almost peculiar to the left side of the heart. Both halves of the heart are equally exposed to the affection in question, from insufficiency of the valves, which though not opposing any hinderance to the natural course of the blood, occasions inevitably its reflux. It is obvious that ossification must lead more readily to hypertrophy, insufficiency of the valves to dilatation, where the substance of the heart is, in other respects, healthy, and where there exists, in both instances, an equal degree of vital power—of irritability of the heart.

*Narrowness* of the orifices, independent of valvular defect, was recognized by Meckel (Mém. de Berlin, vol. xii, obs. 17,) and by Andral (Clin. Méd. 4me edition, vol. iii, p. 58,) as a congenital vice; and, to the best of my knowledge, it occurs in the aorta and pulmonary artery only. Narrowness of the origin, and partly also of the course of these vessels is sometimes the sole cause of the hypertrophy met with in young persons, especially of the male sex, after the age of puberty, and which proves fatal before maturity is attained. Whether, as supposed by some authors, a faulty development of the peripheral system of vessels can induce augmented volume of the heart, I will not undertake to decide; although this view might throw some light upon a case where, in a male 22 years old, I found hypertrophy, of no slight extent, and otherwise unaccountable, coupled with striking diminutiveness of the liver, spleen, and kidneys. At all events these relations merit further consideration. Diseases of the parenchyma of the lungs, involving oppressed circulation through the pulmonary artery, are eminently influential as a source of these affections of the heart, especially of its right portion. Of this tendency are the collapse which occurs in a large portion of the lung during the slow progress of tubercular disease, more especially after the latter has gone through the healing process; obliteration of the texture of the lung, which, under various circumstances, precedes bronchial enlargement; and, above all, emphysema of the lung. (See the respective chapters.) The usual consequence of these morbid conditions is hypertrophy with dilatation of the right heart, to which a modified dilatation, or hypertrophy with dilatation of the left occasionally supervenes.

*Preternatural width of the arterial orifices,* which is reckoned amongst the *causes,* is probably only a *consequence* of partial hypertrophy with dilatation. It is always present, except in the instances before mentioned, and in those referrible to faulty valves; hence the mouth of the aorta is found much wider than that of the pulmonary artery, when the left side of the heart is affected, and conversely. It ought not to be overlooked, that in very aged persons, and in the case of atheromatous deposits occurring in the ascending aorta, the caliber of this artery, at its origin, may, even without hypertrophy or dilatation of the left ventricle, equal, or exceed that of the pulmonary artery.

*Dilatation,* whether simple or *with attenuation,*—a complication in a great measure peculiar to the right chambers of the heart,—is almost always contemporaneous with dilatation of the connecting veins and with phlebectasis generally. Whether this be the cause or the consequence of the heart's dilatation is not known. In most instances, both probably depend upon one and the same morbid condition; as such Lobstein enumerates the hemorrhoidal predisposition, but especially menstrual irregularities, which might serve to account for simple dilatation of the right side of the heart being more frequent in women than in men. *In phthisis,* passive dilatation of the right auricle, and sometimes even of the right ventricle, is not uncommon. We must, however, guard against mistaking an apparent for a real dilatation; since, where death is occasioned by impermeability of the pulmonary substance, those cavaties are commonly gorged with blood,—or if death be preceded by an inflammatory state, filled with fibrinous coagula.

*Hepatic* disorders effect inconsiderable passive dilatation in the instance of fatty heart only; they engender, besides, a thickening, only ascertained by measurement, in the walls of the right auricle and ventricle. Thus, in my tables I find almost invariably in the cases presenting the largest dimensions, short of actual hypertrophy, hepatic disease noted; and it appears that enlargement resulting from chronic inflammation, with or without fatty or wax-like degeneration, bears a much closer relation to diseased heart than cirrhosis and simple fatty degeneration. Liver disease may, however, be a consequence, as well as a cause of disease of the heart. We rarely meet with disease of the heart, coupled with stagnation of blood in the right chambers and in the veins generally, without hyperæmia, and a flabby condition of the liver. Nay, this stagnation may go so far as to occasion extravasation of blood within the parenchymatous texture (Bouillaud). The so-called nutmeg-liver is a very common sequence of hypertrophy and dilatation of the heart.

It is therefore clear that various morbid processes contribute, some singly, others collectively, towards the development of the different forms of hypertrophy and dilatation; and although they cannot always be traced to inflammatory action, it must be confessed that this, in many instances, not only manifests its influence during life, but is discoverable after death. *Endocarditis* is more particularly the occasion of hypertrophy with dilatation of the left side of the heart,—partly from its products narrowing the orifices, or disqualifying the valves, —partly from the innermost muscular layers being more or less paralysed by the adjacent inflammation. The former circumstance causes hypertrophy, the latter dilatation. Thus in persons suffering from endocarditis and not recovering, but dying some time afterwards with augmentation of all the symptoms of heart affection, we find the heart to a certain degree hypertrophied or dilated. For similar reasons inflammation of the muscular substance of the heart determines, in like manner, general or partial dilatation; we then frequently here and there find the well-known residue of antecedent muscular carditis. *Pericarditis* also frequently induces paralysis of the contiguous muscular layers, and consequently dilatation, the degree of which is commensurate with the violence of the inflammation, and the liability of its products to become organized. The consecutive dilatation often extends beyond the heart to the commencement of the great vessels, the contractile layers of their parietes becoming palsied. Even remote inflammation may exert a decided influence upon the origin and disproportionate development of these heart affections. In this manner slight cases of hypertrophy, having perhaps existed for years, and manifested trivial symptoms perhaps at intervals, will, on the occasion of some external injury, or of a pleuritic or other inflammatory attack, make rapid strides and prove fatal within a short period. *Rheumatism* and *gout* may likewise bring on hypertrophy of the heart directly, although, more usually, indirectly, through atheromatous deposition. Hence we so frequently meet with adhesions of the lungs to the pleura and pericardium, or of the left lobe of the liver, of the spleen, &c., to the peritoneal coating of the diaphragm, and finally with vestiges of plastic effusion within the pericardium, such as milk-spots, or partial and general adhesions of the heart, in conjunction with hypertrophy.

The male *sex* and advanced *age* are most prone to hypertrophy. Out of 39 cases, 17 apply to women, 22 to men; 16 to persons below, 23, beyond the age of 40. It is a remarkable fact, that hypertrophy is not merely frequent in stout, powerful, muscular individuals of both sexes, but also in those afflicted with lateral curvature of the spine. According to Rokitansky,[1] very marked hypertrophy and dilatation are scarcely ever concurrent with tubercular disease.

Hypertrophy of the heart induces, more or less directly, many consecutive diseases in the rest of the organism. The most constant results of disturbed circulation are *serous collections* in the cellular texture, especially of the extremities, and also within the serous cavities. Next in frequency is *capillary stagnation* within parenchymatous and membranous organs. Thus in the respiratory organs, and still more in the intestines, we find the veins of the submucous tissue within a greater or smaller compass gorged with blood; and the resulting stagnation in the portal system, evinced in the tense and turgid spleen, eventually extends to the liver, as already pointed out. In many instances the stagnation ultimately determines rupture of the capillaries, producing hemoptysis, epistaxis, menorrhagia, apoplexy of the lung, liver, brain, &c. In the kidneys congestion is well marked; the cortical substance being often more largely developed than usual, of a dark gray colour, and highly injected. The lungs sympathise more or less, partly owing to the general disturbance in the circulation, partly to the mechanical pressure from the enlarged heart. In several instances of considerable hypertrophy, I have seen the resulting pulmonary affection merge in inflammation. The inferior lobe, sometimes of the left, sometimes of both lungs, was displaced, shrunken, not crepitant, softened within a greater or smaller range, of a clear reddish brown colour, and of augmented density. On being cut into, the section was tolerably even, presenting only here and there those soft elevations which, in pneumonia, produce the well-known granular aspect; these, when aggregated in distinct groups (which was however seldom the case) were moister, more decidedly gray (if not of a dirty yellow), and much softer than the rest of the texture. While the whole remainder of the lungs was marked by an excess of blood, the portions alluded to contained more or less of a dingy reddish, troubled fluid; circumstances all combining to establish a peculiar modification of the second stage of inflammation of the pulmonary substance.

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[1] Oesterr. Jahrb. vol. xxvi.

The last point is the relation of hypertrophy to *apoplexy* of the brain and of the lungs. The first is a frequent consequence where the left, the second where the right ventricle is concerned;[1] and in either case the influence of augmented activity of the implicated portions of the heart's muscle, in determining apoplexy, is very naturally and generally admitted. Rochoux[2] combated this doctrine in the instance of cerebral apoplexy, which he held to be invariably due to previous softening of the brain. It is unquestionably true that many cases of apoplexy occur independently of hypertrophy; nevertheless heart disease must be looked upon as conducive to seizures of this kind. Bouillaud considers ossification, or general atheromatous degeneration of its arteries, as disposing causes in the brain.

The inflammation and atrophy of the eyes, as well as the gangrene of the extremities, alleged to result from hypertrophy of the heart, are not confirmed by experience, although they have become traditional, so to speak, through repeated citation from Testa and Corvisart.

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[1] In 57 cases of hypertrophy of the heart, Bouillaud met with cerebral apoplexy six times, pulmonary thrice. I myself met with the former twice, the latter once, in 11 cases.

[2] Arch. gén., 2me sér., vol. xi, p. 167.

### SECT. VIII.—FATTY CONDITION OF THE HEART.

Of this affection two forms may be distinguished; namely, excessive accumulation of fat in the cellular tissue betwixt the serous investment and the muscular substance of the heart, and fatty degeneration or infiltration of the muscular substance itself.

In the natural state there is, even in the adult, but little fat beneath the serous investment. That little occupies both sides of the transverse groove at the heart's base, taking the track of the coronary vessels as far down as the apex,—gradually decreasing in quantity, and being always more abundant on the right than in the left ventricle. Where the fat is in excess, the right heart, in particular, becomes loaded,—its borders down to the apex first, and next its whole anterior surface taking on a layer, more or less deep, of fat-cells. This is not a consequence of general hypertrophy of the heart, but, on the contrary, almost always associated with attenuation and flabbiness of the walls. The adipose substance itself differs from the ordinary type, being less consistent, more oleaginous, and often of a dingy, dark yellow hue.

In the progress of this morbid accumulation, the fat-globules collect not only within the compartments of the subserous cellular tissue, but are freely deposited within the muscular substance likewise, and even between its primitive fibres. This deposition proceeds from without inwards, and insinuates itself immediately under the inner surface of the heart, and betwixt the fibres of the papillary muscles. It is associated with softening and decoloration of the muscular fibres, which present the yellowish aspect of fallen leaves, create grease spots when pressed upon paper, and are easily torn or crushed. The walls of the right ventricle (of the left rarely, and in minor degree) are especially prone to this degeneration, which, though mostly confined to the apex of the heart, often spreads either along its sharp edge, or else towards the pulmonary artery, and in rare instances [1] involves the greater portion of the right ventricle.

This morbid state, so far from being merely local, as some pathologists believe, is the result of various affections in other organs; and though not manifested by any specific local symptoms, its presence may nevertheless be inferred, with tolerable certainty, from collective symptoms referrible to other parts of the body. Thus, in functional disturbance of the larger organs of secretion, and especially of those engaged in the elaboration of venous blood, we meet with fatty encumbrance of the heart in its second stage; the anormal condition being then conjoined with other changes, all dependent, more or less directly, upon hepatic or pulmonary disease, or at any rate indicative of venous plethora. In 13 cases of fatty degeneration of the muscular substance, in its second stage, I found upon cadaveric inspection, the liver invariably diseased, being six times in the granular, and thrice in the fatty state. In 7 cases there was deposition, more or less considerable, of blackened masses (cicatrized tubercular cavities), and in 4, actual tubercle in the lungs; in 8 cases, hemorrhoidal and vesical phlebectasis, in 3, varicose veins of the leg. Under such circumstances, the heart often appears more prone to fatty infiltration than the subcutaneous cellular texture, which, in 4 of the above cases, was nearly devoid of fat. Bizot makes the same observation in reference to the first stage; in 17 cases out of 59 in which the fat had almost totally disappeared from beneath the cutis, it was deposited, more or less copiously, upon the heart's surface. Fatty infiltration of the liver generally implies that of the heart also, although the converse does not hold. Albers erroneously maintains that atheromatous disease is never coincident with this affection; R. W. Smith observed the contrary, for in 7 of 13 marked instances of fatty heart, the arteries presented the most varied atheromatous changes.

*Advanced age* appears to exert a material influence over the affection, owing probably to diminished activity of the skin, coupled with senile atrophy of the lungs. The subjects of the above 13 cases were, in two instances only, under 40, in six, beyond 70 years of age.

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[1] R. W. Smith, Dublin Journ. July, 1836.

The influence of *sex* is still more remarkable. Amongst 35 males Bizot found but 4, amongst 42 females no fewer than 23, affected with fatty encumbrance of the heart's surface. Nor did this appear to be connected with the greater tendency of the female sex generally to obesity; for of those 42 individuals, 29 were quite emaciated, and of these latter 14 exhibited the disease, whilst amongst 30 emaciated men this was the case with only 3.

The pathological relations before alluded to may, through increased disturbance and continued retention of the secretion, cause the fat not only to accumulate, in an inordinate degree, upon the surface or in the interior of the body and of its organs, but also in *the blood itself,* in the form of oil, as detected in two instances by R. W. Smith (l. c.), and in another by myself. This fact appears to gain in importance in a pathological point of view, when we reflect upon the extraordinary rapidity with which dead bodies thus overcharged with oily substances undergo putrefactive, and especially gaseous decomposition; and that a morbid development of fat has in many instances its remote cause in the abuse of spirituous liquors,—a view corroborated by medical experience. Might not these facts serve to throw some light upon the intricate question of spontaneous combustion? [1]

It might appear that the softening attendant upon fatty infiltration of the heart, must render it prone to rupture. The fact, however, of rupture being almost exclusively restricted to the left side of the heart, is opposed to the assumption, *a priori;* and in truth there are not more than four definite cases of the rupture of fatty heart upon record (R. Adam, Bouillaud, Albers, Smith).

Rokitansky has described a third perfectly distinct species of fatty heart, occurring almost exclusively in hypertrophied hearts, which at the same time exhibit the remains of earlier endocarditis and carditis. In this form the fat does not accumulate in masses, there being no fat-vesicles inclosed within fasciculi of cellular tissue, but is beaded, as it were, in minute microscopic granules, closely interlaced, and imbedded among the primitive fibres of the heart's muscles. Those primitive fibres have lost their transverse striæ; the fibrils are friable, and easily reduced to minute molecules. The whole heart may suffer this species of fatty infiltration, although commonly distinct portions alone are diseased. In the latter case the flesh is here and there found, within a certain sphere, pallid and dull, of a dingy yellow,—soft and flaccid; at a more advanced stage these spheres become more numerous, and pass very gradually into the healthy mass; the endocardium has become thin and transparent, rendering the dull aspect of the heart's muscle here and there conspicuous; the trabeculæ, and the papillary muscles are either wholly or partially diseased. In its greatest extent this fatty condition may affect an entire ventricle, more commonly the left, although, when partaking of the hypertrophy, the right ventricle, and consequently, as above stated, the whole heart, may become involved. Rokitansky is of opinion that spontaneous rupture of the hypertrophied left ventricle is, in the majority of cases, referrible to this disease of the heart's muscle.

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[1] See, with reference to this point, Smith's first case.

In youth this form of fatty heart has been observed, even where there was no hypertrophy, the organ being dilated, probably in consequence of the degeneration. Rokitansky believes that, when in such instances the papillary muscles are diseased, the consequent imperfect tension might occasion insufficiency of the valves.

## SECT. IX.—CYANOSIS. MORBUS CŒRULEUS.

Of late years it has been very generally admitted that the cyanotic state is not symptomatic of one, but of various diseases, both of the lungs and heart. Nevertheless, for brevity's sake, we shall here use the term *cyanosis* in its older and familiar acceptation, namely, as denoting a preternatural communication between the right and left compartments of the heart.

This morbid condition is for the most part dependent upon congenital malformation; as such, however, it is (as clearly demonstrated by J. F. Meckel) not to be regarded as merely accidental, but as subject to the same laws which govern the normal development of the organism.

A preternatural communication between the two chambers of the heart, and the commingling of arterial and venous blood sometimes consequent thereupon, occurs under a great variety of circumstances. In the following pages, however, our attention will be devoted almost exclusively to those forms in which life is capable of considerable prolongation subsequently to birth.

The first in importance is an unclosed state of the foramen ovale,—a malformation not only the most frequent of those we have to consider, but at the same time almost invariably a complication of all the rest. Incomplete closure of this aperture, admitting, through an orifice at its margin, the passage of a probe or of the handle of a scalpel, in short, imperfect *valvula foraminis ovalis,* is very often unattended by cyanotic symptoms. Out of 155 individuals, none of whom had manifested any such symptoms, Bizot found the foramen ovale more or less open in 44 instances.

Where, however, the aperture remains destitute of either a sufficient muscular plug, or an adequate valve, secondary disease of the heart, eventually productive of permanent, or at least of fleeting symptoms of cyanosis, has been invariably known to follow.[1] The marginal edges of the unclosed *foramen ovale* are partly tendinous, partly muscular, sometimes almost of callous hardness. In certain instances the valve or the fleshy plug is variously perforated, the width of the aperture varying from 1½ to 15 Parisian lines.

The permanence of *Botalli's duct* appears to be rare, and always coincident with unclosed foramen ovale, and with perforation of the septum of the ventricles.

*A communication between the ventricles,* through an aperture in their septum, may coexist with closure of the foramen ovale, and is sooner or later productive of a cyanotic state. Louis has collected 5 cases of the kind (one, that by Jackson, of the English General Whiple); in a sixth related by Otto (Neue seltene Beobacht. p. 49) of a girl aged 12½, the right auricle was wanting, and the veins of the trunk opened at once into the right ventricle. Perforation of the ventricular septum is more frequently observed in common with a patulous state of the foramen ovale and of Botalli's duct; for example, in the instance so often cited from Richerand's 'Physiology,' of a man aged 40, or in Nasse's interesting case of a girl aged 19 (Leichenöffnungen, p. 162).[1] The aperture in the ventricular septum is mostly situate near the base of the heart, immediately below the origin of the pulmonary artery and aorta; sometimes, however, nearer the apex of the heart, as in Otto's case. This corroborates the opinion of Albers, that the perforation is nearer to the base when the foramen ovale is open, and nearer to the apex when it is closed. The communication between the two ventricles is sometimes direct, sometimes oblique,—duct-like. In the case of a child of 13 years old, I once found it crossed by a tendinous band. Its borders are mostly smooth, consisting of fleshy columns; but occasionally, too, of tendinous or cartilaginous hardness. In the cases recorded, the width varies from 2 to 12 lines; it may, however, even amount to partial (Bouillaud,—in a child of 4 years) or almost entire (Burdach,—in a youth of 16) deficiency of the ventricular septum.

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[1] See 9 cases collected or described, partly by Louis (Mém. Anatomico-pathol. p. 301), partly by Seiler (Horn's Arch. vol. viii, p. 805), partly by Otto (Seltene Beobachtungen, p. 96), partly by Meckel (Path. Anat. vol. i, p. 447), of cyanotic persons between the ages of 18 and 60. (Meckel.)

The cardiac origin of the great arteries, is liable in like manner to many anomalies, all attended with malformations of other kinds within the heart, mostly proving fatal at an early period under the symptoms of cyanosis, and seldom compatible with prolonged life. In five cases collected by Albers, the pulmonary artery sprung from the left, the aorta from the right ventricle; in all, death ensued within a few months after birth; the two eldest of the children having respectively attained the age of three (Albers) and five (Farre) months. An interesting case is related by Martin, (Müller's Arch. 1839, fasc. 3, p. 222,) in which death occurred after the tenth week. In this state mixed blood circulates, occasioned by the open condition either of the foramen ovale, or of Botalli's duct, or else by an aperture in the ventricular septum.—In two cases of Farre's, (an infant of three weeks, and a male child of eight months,) the pulmonary artery arose from both ventricles. The proper aorta gave off the arteries of the head and of the upper extremities only, and united by a thin branch with the descending aorta, which was derived from the pulmonary artery, beyond the point where the latter sent off its branches to the lungs. The origin of the aorta from both ventricles has been more frequently observed. In seven instances (Louis, Albers, Bouillaud, and Meyer) life was long protracted. The most remarkable examples are, of a girl of 9 years (Cerutti), a boy of 13 (Sandifort), a girl of 17 (Albers), and a woman of 25 (Tommasini); a girl of 17, admirably described by Meyer (Rust's Magazine, vol. lv, fasc. 1, p. 158). In this malformation the pulmonary artery is mostly very narrow, and its semi-lunar valves, like those of the aorta, are either partially or wholly wanting, being faultily fashioned, or rudimental only.[1]

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[1] These examples confute the notion of Albers, that a continuance of life under such circumstances is impossible.

Are the preternatural communications between the right and left divisions of the heart, just described as the source of cyanosis, always to be regarded as *congenital* malformations? Louis answers this question in the affirmative, and as far as concerns the anormal origin of the great arteries, the coincidence of perforation of the heart's walls with a pervious state of Botalli's duct, and with other malformations acknowledged to be congenital, the thing is indisputable.

It is otherwise, however, with respect to simple perforation of the ventricular or auricular septa. Bouillaud holds these to be in part the result of ulcerous destruction of the muscular substance with subsequent cicatrization, and refers in particular to a case of Thibert, in which the edges of a perforation of the ventricular septum, in a man aged 24, were furnished with a yellow membranous fringe. There is, however, no mention made of softening of the vicinity, or of other changes of the endocardium, usual in ulcer of the heart; moreover, the foramen ovale was open, and precisely in the condition in which it is wont to be found in a malformation obviously congenital. Meckel and Abernethy believe in the possibility of the foramen ovale *reopening* in advanced age,—especially in disease of the lung. Otto, who seems of the same opinion, adduces 13 cases of dropsical accumulation within the pleural sacs, with strong adhesion, induration, inflammation, or suppuration of the lungs, (in one instance with coarctation of the pulmonary artery, in another with ossification of the pulmonary sigmoid valves,) in which the foramen ovale was found open; cyanotic symptoms having for a longer or shorter period preceded death. He particularly refers to the case of a man of sixty who had died of hydrothorax, and in whom the right side of the heart and the pulmonary artery were found dilated; the foramen ovale being indeed closed, but its fossa of such depth as to project like a thin pouch into the left auricle. He looks upon this example as demonstrating the way in which gradual reopening of the foramen ovale may take place. It must, however, be borne in mind that the unclosed state of the foramen ovale may, in several of the 13 cases, and especially in those in which the pulmonary artery deviated from natural, have existed from birth, without betraying itself by any decided symptoms, until some disease in the respiratory and circulating organs supervened; and, again, that the cyanotic symptoms may have sprung from the pulmonary affection alone, imperfect closure of the valve of the foramen ovale (which condition Bizot[1] has shown to be no less frequent than insignificant) being merely a coincidence. In a word, although the possibility of acquired perforation of the heart's septa cannot be denied, no positive example of it has been witnessed.

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[1] The common origin of the aorta and pulmonary artery from either ventricle alone has not been sufficiently illustrated by examples. The present account of cyanosis has been restricted to cases in which life had lasted for some time. Further information respecting cyanosis or malformations of the heart is furnished by Gintrac (sur la Cyanose, 1824), by Albers (Erläuter. p. 140 et seq. and Abtheil iii, pl. xii–xviii of the Atlas), and especially by Meckel, in his Patholog. Anat.

The relation of the *Eustachian valve* to the unclosed foramen ovale, first hinted at by Wolf, and more closely investigated by Meckel, is not unimportant. Among 80 hearts examined with reference to this point, he found 43 instances of open foramen ovale, with the Eustachian valve large, often muscular, and entire, or but slightly reticulated; in a woman of sixty in particular, the foramen ovale was an inch wide, and the Eustachian valve two inches long, almost half an inch high, and very strong. In 16, the foramen ovale was closed, and the Eustachian valve absent altogether, or merely rudimental, or else torn to pieces. In 13 the Eustachian valve was very strong, often muscular,—and the foramen ovale closed. In 8 there was hardly any Eustachian valve, and the foramen ovale was more or less gaping. The frequency of this favorable condition for the passage of venous blood to the foramen ovale and the left auricle, cannot therefore be doubted; and the presence of a strong Eustachian valve may be regarded as conducive to a mingling of venous with arterial blood.

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[1] It should be added that Bizot has met with this condition of the valve even less frequently in phthisical subjects than in others. Meckel and Otto admit, moreover, that among a great number of persons who had died of pulmonary disease, they very rarely found the foramen ovale open, which statement quite accords with my own experience. I do not, therefore, consider the development of cyanosis from a reopening of the foramen ovale as fully made out by Otto's cases.

But what are the necessary conditions to this morbid commixture of the blood? This question is forced upon us by a consideration of the fact, that the influence of the mixed blood upon nutrition, shall be very palpable in some patients, and null in others. Indeed, in many cyanotic subjects the energy of the vital functions is unimpaired, (females menstruate regularly, and procreate healthy children, &c.) while others are constantly ailing, highly sensitive with respect to cold and other external influences, stunted in growth, (many emaciate, others again prone to obesity,) and perish, either early in life, or at one of the periods of evolution. In the former instances, therefore, the passage of venous blood into the arterial, must either not occur at all, or but to a slight extent, or only at certain periods. Jules Cloquet and Louis have shown that, apart from the cases in which a preternatural origin of the blood-vessels necessarily keeps up an incessant mingling of the two kinds of blood, it does not take place through an orifice in the septa of the heart, when the walls of the two cavities which communicate with each other, are of equal thickness and strength, and scarcely, even where their strength is unequal, provided the heart's orifices are sufficiently ample. And in reality, wherever there is impaired nutrition, those relations are found wanting in various ways. In most cases there is present hypertrophy of the right auricle and ventricle, mostly eccentrical, but sometimes also concentrical. The walls of the right ventricle are frequently much thicker than those of the left, whence the heart assumes an almost globular shape, and quite a transverse position. Sometimes the left auricle and ventricle are simultaneously hypertrophied, though in not more than a fifth of the cases. The heart is then enormously enlarged, so as to embarrass the neighbouring organs more or less, and to deviate from its natural position both behind and below. Under these circumstances, inflammatory phenomena about the heart and pericardium are not unfrequent precursors of death. In six cases described by various authors, the signs of pericarditis, though for the most part of a modified character, were cognisable.

*Coarctation* of the *right arterial orifice,* which is observed, in a smaller or greater degree, in about one half of the cases, must here exert a most material influence. On some occasions the pulmonary artery, at its commencement, is considerably narrower than usual, and does not dilate, until after the reception of Botalli's duct, in its pervious state ; or there are ossifications at its valves two of which perhaps are alone present ; or in place of the valves there is a transverse membranous expansion, with a minute aperture ;[1] or the approach to the pulmonary artery within the ventricle, is contracted, forming a small channel, or, as in Nasse's case, blocked up by fleshy columns, hardly separate from each other. In all these instances, it is obvious, that the venous blood is more or less readily impelled directly into the left division of the heart. Nevertheless, there being not rarely a concomitant coarctation of the right auricular orifice, whether simple or arising from preternatural formation, or from ossification of the tricuspid valve, the possibility of arterial blood passing into the right side of the heart, especially during diastole, must be admitted,— more particularly where the right chambers of the heart are at the same time dilated. Upon the whole, this last mode of preternatural admixture of the blood must be acknowledged to be highly influential in determining the hypertrophy and ossifications of the right side of the heart, so common in cyanosis ; inasmuch as these morbid conditions are otherwise almost entirely restricted to the left side, which *in cyanosis* is for the most part exempt from them.

The most remarkable symptom of the organic conditions described, namely, the *blueness of the skin,* was formerly thought to be amply accounted for by the mingling of arterial with venous blood: the simple fact, however, that the decoloration is not permanent throughout life, deprives the explanation of all its weight. The cyanotic phenomena sometimes do not set in until the third, seventh, or fourteenth year, nay, even later, —it may be not until shortly before death; occur periodically only, and at long intervals; are frequently brought on by quick walking, hasty movements, passionate emotions, intercurrent diseases, (hooping-cough, for instance,) external violence, and the like, and thus become permanently established, or else again vanish for a time. If the blueness depended upon the character of the blood, these alternations could not subsist. When, moreover, we reflect that in the fœtus, in which mixed blood always circulates, no such decoloration is perceptible; and that in a case by Breschet, in which the left subclavian arose from the pulmonary artery, no alteration of colour was visible in the left arm; we feel it necessary to look for other grounds for an explanation. This was already indicated by Morgagni. Kreysig (Herzkrankheiten, vol. ii) was, however, the first to form a correct view of the subject, and Louis has subsequently demonstrated that the cyanotic tinge results from impeded flow of the venous blood back to the heart, or from thence to the lungs; any obstacle to the entrance of the blood into the right ventricle, or to its exit from thence into the pulmonary artery, being of course, in a high degree, propitious to such stagnation. It may be inferred from the foregoing, that from the organic relations peculiar to cyanosis, the stagnation in question may increase vastly on the preexisting impediments to the circulation being augmented by accidental causes, whether external or internal.

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[1] Lediberder found the pulmonary artery in an infant, 12 days old, closed by a membrane of this kind, without any aperture whatever. (Bullet. de la Soc. Anat. t. ii, p.68.)

Among the morbid phenomena in other parts of the organism, incident to cyanosis, there is not one that is not common to disease of the heart generally, and to hypertrophy in particular. Even the bulbous shape of the distal phalanges of the fingers, and the incurvation of the nails, are no more peculiar to cyanosis than to sundry pulmonary diseases. A curious circumstance is the alleged tendency to whitlow among cyanotic individuals. Diminutiveness of the spleen, thyroid gland, and renal capsules, as urged by Nasse, is by no means constant, the reverse having been found in several instances.

# PART SECOND.

# DISEASES
OF THE
ORGANS OF RESPIRATION.

# PART II.

## CHAPTER I.

### DISEASE OF THE PLEURAL MEMBRANE.—PLEURISY.

In no part of the organism are the general phenomena, and especially the products of inflammation, more strongly marked than upon the free surface of the pleura, where we meet with fluid and solid effusions of every gradation and variety, whether as regards the intensity or duration of the disease, changes affecting the latter during its progress, or peculiarities dependent upon cachectic influence. Nor do we fail to recognize a different relation of the inflammatory process, in different portions of the pleura. Thus, plastic and liquid effusions are more copious upon the diaphragmatic, and upon the inferior surface of the costal pleura, whilst in the upper half, the inflammatory action becomes less and less intense, the nearer the apex is approached. The pulmonary pleura is least prone to copious, and especially to fluid effusion, except where it is reflected between the lobes and about the root of the lungs, beyond the entrance of the great vessels and bronchial tubes. In these situations there is a stronger layer of cellular tissue beneath the pleura, whilst the vicinity of the bronchial arteries determines thither a greater abundance of blood-vessels.

Pleurisy is a disease of great frequency. It may extend over the greater portion of one or of both pleural cavities, or be confined to small isolated patches. It may occur idiopathically, or be associated with acute and chronic affections of neighbouring organs. Sometimes it sets in amid symptoms the most violent and oppressive, but disproportionate alike to the speedy remission, the trivial character and insignificance of the morbid product, and the physical signs. In other cases the effusion may prove perilous or fatal through its character and amount, without, for a time at least, giving rise to general symptoms sufficient of themselves to excite much alarm. Few subjects are examined without presenting some vestiges of former pleuritic affections. These consist in adhesions betwixt the pulmonary and the costal pleura, and are so frequent that physicians of old did not regard them as preternatural; nor do many, at the present day, consider them as necessarily connected with inflammation. This opinion is founded upon the fact of these adhesions being met with in individuals not known to have ever suffered from any inflammatory affection of the chest. But until satisfactorily traced to some other cause, it would appear more proper to refer them exclusively to an inflammatory origin. Such adhesions may assuredly become developed without any manifestation of disease sufficiently striking to impress itself upon the patient's memory. It may be added, that the practitioner has need of peculiar tact and perseverance in directing the attention of a careless, and perhaps unwilling patient, from present to bygone ailments. Again, how easily may a pleurodynia, a rheumatic affection of the thorax, attended only with fleeting pains, perhaps scarcely sufficing to confine the patient to bed for a few days, engender even during so brief a period a thin layer of organizable effusion between the costal and pulmonary pleura. Upon the smooth and free surface of the pleura, the slightest inflammatory action is wont to cause a deposition of lymph, which, incapable of being expectorated, like matter forming upon the mucous surface, has to suffer ulterior changes within a shut sac. This fact, that in the inflammation of serous sacs, the morbid products find no natural vent, and are altogether dependent upon the assimilative powers of their matrix, is of the highest import, and alone sufficient to show how greatly the duration of the disease must, irrespectively of dynamical causes, be modified by the degree of the inflammation and the corresponding character of the effusion. For, as was observed in reference to pericarditis, in serous membranes inflammation becomes chronic, only when there is originally engendered a product incapable of being either absorbed or organized. The degree of inflammation determines the character of its product, and this the whole progress and issue of the disease. Here it may be observed that, in the inflammation of serous membranes, Nature points, so to speak, the attention both of the patient and of the practitioner, to the momentous character of the incipient stage, by the violence of the local symptoms. Attention, thus aroused, should not, however, be allowed to slumber again, should there be an early remission of the pain; for, in pleurisy, no less than in pericarditis, pathological anatomy has taught us the grave fact, that the most copious and perilous effusion frequently takes place precisely during such deceptive remission.

The first appearances of inflammation of the pleura consist in a congested state of its blood-vessels, which are seen congregated, here and there, in dense though delicate nets, beneath the still transparent membrane. At certain points the bright red colour deepens and becomes more equalized; these points are somewhat prominent, and though scattered at first, presently crowd together and get encompassed with a progressively enlarging zone of gorged blood-vessels. At the same time patches and streaks are observed either darker than the rest, and not unlike little ecchymoses, or else of a pale red hue, as if from imbibition. The pleura now speedily loses its smoothness and polish, becoming dull, and looking, as Laennec expresses it, as if daubed over with a paint-brush. This redness gradually spreads until, in most instances, the whole, says Gendrin[1], becomes uniform. The first rudiments of an adventitious membrane now become perceptible, the spots originally reddened, and that chiefly by repletion of the vessels, presenting little dull white or yellowish points, which rise above the serous surface in the shape of flat granules, and ultimately coalesce.[2]

During the above proceeding in the serous membrane, the deeper seated layers of cellular texture subjacent to the pleura, intermediate between the fasciculi of the intercostal muscles, and even external to the chest, are distinguished everywhere near the part affected by great vascularity, together with punctiform ecchymosis; the meshes or intervals between the cells being here and there filled with a yellowish, half-fluid, half-gelatinous effusion. This implication of the external cellular texture, for the most part only apparent at the commencement of the disease, is by no means a constant phenomenon, and becomes less and less perceptible as the disease advances. In rare instances the two affections, that of the external cellular texture and of the pleura, keep pace with each other; and purulent formation, lardaceous thickening, or else serous infiltration and softening, as the case may be, are often extensively diffused.

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[1] Description anatomique de l'inflammation.

[2] In speaking of the blood-vessels, the existence of which was disputed until lately, it is right to advert to the recent researches of Henle on the structure of serous tissue. According to that physiologist, this tissue consists of several layers of superimposed cellular tissue, more and more closely attached to each other, the free inner surface being a thin layer of epithelium-cells. There are blood-vessels in all the layers except in this exceedingly delicate epithelium-membrane; the inflammation does not therefore proceed from the epithelium, but from the subjacent cellular tissue. The epithelium seems to be thrown off at the outset. It is to be regretted that microscopic research has not been extended to the pathological relations of these structures described by Henle.

The attempt has been made to reckon the progress of pleurisy by several defined stages, as follows: First, the period of dry inflammation, which is of shorter or longer duration and comprehends all the changes in the pleura and sub-serous cellular texture above enumerated, being further characterized, as is supposed, by a total suppression of serous secretion. I have never encountered this dry stage as described; having always, even at the very outset, found the serous fluid somewhat, however slightly, augmented in quantity, and marked by its deep yellow tinge and its increased consistency. There were, likewise, almost invariably present those grayish or yellowish points, the initial and quickly expanding rudiments of membranaceous effusion. The so-called *pleurésies sèches* of Andral (Clin. Méd. 4me ed. vol. iv, p. 405) are therefore probably to be understood in a comparative sense only—effusion too scanty to be detected by physical signs during life, being counted as nothing compared with the amount contained in other cases. Such a ground of distinction is, however, obviously opposed to the strict principle of pathological anatomy. The second stage is eminently that of effusion, which, although not rigorously speaking confined to any one period, is not sufficiently copious and characteristic to constitute a secondary and distinctive stage, until the original inflammation has become thoroughly developed. To divide the period of effusion into two stages, and thus attempt to discriminate between the development of fluid effusion on the one hand, and of coagulable lymph and adventitious membranes on the other, is discordant with the process which gives rise to those formations. The third period would be that of organization of the plastic exudation. This, however, does not take place at any particular stage, nor is it a process to which every form of plastic effusion is necessarily subject. It would be difficult to reconcile, under the above classification, those pleuritic products which exhibit traces of organic deviation after the first twenty hours, and those on which the power of assimilation has been ineffectually expended for weeks or months.

Whilst in some instances, therefore, this division would seem to correspond tolerably well with the natural course of events, in others it is wholly inapplicable. The attempt might perhaps answer better, to consider the secondary processes connected with pleurisy according to the various degrees of intensity with which they ensue upon the primary act of inflammation.

Where the progress of the inflammation is soon arrested, the product is inconsiderable. So far as the vascular formation extends, the pleura is found invested with a very thin layer of plastic exudation ; and this delicate membrane, which is mostly opaque, often veils the inflammatory redness so thoroughly, that it requires a practised eye to detect at once the traces of pleuritic inflammation. The liquid effusion contained in the pleural sac consists either of a small quantity of a yellowish limpid fluid, or (where the inflammation commences amid the phenomena of *hypercrisis*) of a more copious collection of slightly troubled, reddish, or mahogany-coloured serum, pervaded by delicate membranaceous flocculi. Upon the condition of the thin adventitious membranes alluded to, depends the length of time requisite for the absorption of the fluid. The more heterogeneous the quality of the former, the greater will be the impediment to the absorbents acting upon the latter. However, they can scarcely oppose any abiding obstacle, since for the most part they either enter into organic union with the serous substratum, or else dissolve piecemeal in the fluid.

Where, on the contrary, the inflammation maintains itself at its original height, or either suddenly, or else gradually and more or less steadily increases, the morbid product accumulates in like proportion, exhibiting at the same time, for reasons not ascertained, the most manifold differences.[1]

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[1] This variety of character would appear to be entirely founded upon individuality. If not, why should the same morbid action bring about such dissimilar results in different persons, under the identical outward circumstances, and not only in man

The product most simple and least embarrassing to the organism in its ulterior progress, though capable, where the inflammation is at all intense, of very rapid development, is an almost transparent yellowish *jelly*. This is partly diffused in layers between the costal and the pulmonary pleura, partly subsides to the lowest portions of the pleural sac in flakes or pellets, surrounded with a small quantity of fluid effusion. This gelatinous substance consists almost exclusively, and in equal proportions, of the fibrin and the serum of the blood, with a little colouring matter, attached here and there to the surface. It is susceptible of organization throughout, and blood-vessels form in it with surprising rapidity. Andral found in rabbits, after not more than nineteen hours, numerous arborescent reddish lines crossing it in various directions (Clin. Méd. vol. iv, p. 538). In a case of phthisis, in which pleuritic pains had set in twenty hours previous to death, I met at the corresponding part, this species of jelly, in which delicate blood-vessels were observed shooting from the borders. This product soon affixes itself to the surfaces of the serous membrane, wherever they approach each other, and a few days appear sufficient to effect a tolerably extensive coalition between the two. The rapid growth of vessels gradually confirms this bond of union, and the aqueous parts soon becoming absorbed, nothing at length intervenes between the two formerly separate membranes, except a soft, highly vascular, cellular layer. The disease thus terminates very gently, without the function of the affected part undergoing any abiding or material disturbance. Only where the inflammation, after having been almost extinguished, happens to become rekindled, the incipient adhesions may become again dissolved, or else retarded or entirely arrested by other morbid deposits.

In pleuritic effusion, however, we do not always meet with substances so thoroughly germane to the organism, or blended together in such happy proportions; ingredients very often enter into its composition which, either from too early consolidation, or from some peculiarity of character, render it less susceptible of organization. These substances are far less easy of assimi-

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but in animals likewise? Andral having excited inflammation of the pleura in several rabbits, on the same occasion, and by precisely the same means, there resulted in some profuse suppuration, in others effusion of organizable matter.

lation, and, acting as foreign bodies, serve meanwhile to embarrass the surrounding parts.[1]

The adventitious membranes, susceptible only *conditionally* of organization, appear not to form so rapidly and suddenly. They consist for the most part of several either quite homogeneous, or else distinctly different layers, largely investing, and rather firmly adherent to the pleura. Their consistence is considerable, resembling that of boiled white of egg, or smoked flesh; they tear easily, and display a fibrous texture. The colour of these plastic masses varies greatly, but is mostly of a dull or yellowish white, and occasionally of a faint red, passing into violet or mahogany colour. This tinge is sometimes equable, sometimes patched and irregular, sometimes again superficial, as if from imbibition. Such adventitious membranes are always opaque, and never translucent. Their surface directed to the pleura is mostly smooth, and corresponds in character to that membrane. On their free surface, facing the cavity of the pleura, they are usually paler and softer, and display somewhat of a net-like or villous fabric. Together with this, the cavity of the pleura usually contains a considerable amount of either a light-brown, or a reddish, or else a slightly troubled and flocculent fluid, bearing, it would seem, a close relation both in quantity and quality to the false membrane itself. The diminution of the fluid is proportionate to the more or less rapid development of blood-vessels in the membranaceous deposit; where, on the other hand, the organization is arrested and imperfect, and the vital energy of the morbid product limited to coagulation, the fluid effusion is relatively more abundant,[2] longer withheld from the influence of absorption,—whilst an increasing proneness to decomposition gradually overpowers those vital changes which would adapt it for the work of reproduction. The above relations lead to a two-fold consequence. First, the disease will become prolonged, and putting on a chronic character not terminate favorably until the adventitious membranes have everywhere become organized. Secondly, the vital effort to surmount the resistance offered by substances so difficult of assimilation, and to effect the absorption of fluids debased by long seclusion, is wont to arouse the sympathies of the whole animal economy; a febrile state passing into hectic supervenes, and a fatal issue menaces, not only on the part of the local affection, but likewise as a consequence of the forcible reaction upon the general health. Absorption of the effused matter is so constantly attended by fever and emaciation, that Hodgkin (l. c. p. 122) regards their absence as evidence of its not taking place. In many instances it does not proceed steadily, but pauses occasionally—sometimes until again called forth by intercurrent disease of another kind.[1]

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[1] It is to be regretted that our knowledge of the chemical constitution of the various forms of effusion is still so imperfect. Whilst it was customary to crowd all of them under the category of plastic lymph, individual analyses, made at hazard upon some adventitious membrane, could lead to no available result, and, least of all, furnish any inference as to the further character of plastic substances with reference to their more or less rapid coagulation or to their organic deviations.

[2] The conditions which regulate the quantity of fluid effused are not fully made out. In older subjects, the most trivial pleuritic seizures frequently lead to a very copious discharge of fluid, although, under circumstances the most favorable to absorption, namely, a trifling amount of plastic substance of a gelatinous kind. In other, for the most part younger individuals, there is,—together with the most abundant plastic, inorganizable exudation,—but a scanty portion of limpid serum, collected, as it were, within separate cells.

When a large proportion of plastic matter is thrown out within a very short space of time, in consequence of violent inflammation, the whole mass often coagulates so rapidly as to become totally unfit to enter into organic union with the parent membrane. It then lies loosely agglutinated to the pleura, as uniformly honey-combed false membranes, or imbricated layers. Sometimes the coagulum is almost shapeless, appearing in irregular flocculent layers, from whence scattered filaments run across the cavity of the pleura, whilst others shoot from the surface, resembling coarse felt or moss. These plastic masses are in general tolerably soft, opaque, and of a pale yellowish hue. A large quantity of fluid is at the same time effused, being often mingled with colouring matter of the blood, which, in such cases, imparts its tinge to the collective inflammatory product. The more solid portions of the effusion surround the fluid like a saccular envelope, which prevents both their direct escape and their immediate reception by the absorbents,—seeing that the adventitious membrane still retains its unorganizable character. The disease thus lingers,—passing, where the inflammation does not subside, into chronic pleurisy,—into hydrothorax, or as some have asserted into empyema, where it does.

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[1] See Andral's 27th case, in which pleuritic effusion remained unabsorbed until during the course of an intermittent fever.

The *purulent* form of effusion may be the result either of a very high degree of inflammation at the outset, or of the introduction of external air reacting upon the inflamed serous surface and its product, during the progress of the disease. Suppuration of the serous membrane is most marked, where pneumothorax with violent inflammation succeeds to the bursting of a pulmonary abscess into the cavity of the pleura. Here the surface of the pleura very soon becomes dull and of a dingy gray,—at the same time secreting a thick, tenacious fluid, which is partly distributed in what resembles flat, very soft granules, partly collects at the base of the serous cavity. Sometimes almost pure liquid pus forms in great abundance; frequently, however, it is mingled in various proportions with coagulable substances, which, as thin, very soft puriform false membranes, become deposited upon, or loosely adhere to the pleura, or else float in the collected fluid as soft flakes or pellets. This form of pleurisy almost always proves rapidly fatal, unless the pus find a ready outlet, or become sequestered within false membranes of sufficient strength. In the latter case (empyema) the patient has been known to survive for a lengthened period, and eventually to perish only through the accompanying hectic fever.

Having examined, one by one, the products of pleurisy according to their leading types, it remains to state, that owing either to different portions of pleura having been inflamed in various degrees, or else to one and the same portion having sustained a succession of inflammatory attacks, alternately violent and gentle, several of these products are frequently met with in the same subject. The most multiform combinations may thus arise, the plastic growths being disposed in superincumbent layers, and the fluids effused either commingling or accumulating by themselves in the intervals between the false membranes. It is easy in anatomical investigation to discriminate betwixt organizable and mere coagulate exudation, as well as between fluids readily and fluids not readily absorbed. Thus is an instructive insight often obtained into the nature of a complex train of symptoms, imperfectly understood during life.

With reference to the mingling of different inflammatory products, it is to be remarked that pus, at whatever period generated, stamps all other substances effused, with its own impress. Plastic organizable effusion is thus liquefied, and converted into pus; that simply coagulated turns yellow, becomes putrescent and flocculent; and in like manner, resolves itself, partially at least, into pus.

Besides the usual forms of pleuritic effusion, we meet occasionally with certain varieties of a nature calculated materially to influence the progress of the disease. The *tubercular constitution* is the most frequent source of such peculiar modification; just as by causing the cytoblastemata of tubercle to become secreted conjointly with the normal elements, it changes the plastic products of nutrition in particular structures even under ordinary circumstances,—so does it exert the same palpable influence upon the formative process when heightened by inflammation. Then are seen (for example in the gelatinous effusion, the most prone to become vitalized, and either simultaneously with incipient development of blood-vessels, or earlier,) scattered dull points which ere long change into so many granules, as big as pins' heads. In the rapidly coagulated false membranes of the second form, tubercle likewise frequently manifests itself in the shape of white, flat granules, perfectly distinct both in consistency and colour from the remainder of the mass, and altogether inaccessible to that vascular development which sooner or later pervades the adjacent membrane. These tubercular granules are cognizable even in the plastic substances deposited from purulent effusion; only in the adventitious membranes, the result of simple coagulation, which are nowise susceptible of organization, have I never met with anything resembling tubercular growth. It is here, like everywhere else, the characteristic of tubercle to prolong the disease, by causing, as a substance alien to the organism, continued irritation, and thus keeping up the secretion of fresh fluid and solid matter; in a word, by permanently contracting or frustrating absorption. In all these cases, tubercles, both old and recent, are present in other organs, and especially the lungs.

Allusion has already been made to an admixture of the accumulated fluid with the colouring matter of the blood. In very rare instances blood itself seems to transude into the cavity of the pleura. This is shown by little gory clots found at the bottom of the collected serum, or deposited between the false membranes; nay, there are examples of the entire effusion consisting of pure blood, and that separated into serum and crassamentum. (See Andral's Clin. Méd., fifteenth case of pleurisy.) Sanguineous coloration of the effused fluid is most frequent in the tubercular form of pleurisy.

The most remarkable of all pleuritic products consists in the exhalation of gaseous substances, a form asserted by some pathologists to be frequent, though called in question by others. Andral (Clin. Méd. vol. iv, p. 517) relates a single instance of the kind, which he appears at first to have considered decisive. In his third edition, however, he observes that the accumulation of air *may* have resulted from perforation consequent upon the softening of tubercle immediately subjacent to the pleura. Nor does this conjecture seem wholly groundless. A case in point, which occurred at the Leipsic hospital, presented exactly similar features; there were here, in like manner, numerous tubercles situate close beneath the pleura, and although no perforation was actually visible, it is by no means impossible that one may have existed, although so minute as to have escaped detection. Further evidence is needed on this head, as likewise touching the possibility of effused fluids becoming decomposed, and giving rise to the development of gases within the shut sac of the pleura, during life. This Hodgkin doubts. In corpses, opened after putrefaction has set in, such gaseous decomposition is sometimes met with.

Upon the whole, the ulterior changes of effused fluids within that confined space are inadequately understood. An opinion formerly prevailed, that adventitious membranes gradually separated from the mere effused fluid, and were converted by a series of consecutive changes into organized growths; the various kinds of plastic products being mere transition-forms of the same exudation.[1] But experiments on animals, and repeated comparison of the course of the disease, with the results of cadaveric inspection, have clearly shown that the character of the effusion varies, from the first, according to the degree of the inflammation and the general symptoms,—that, in each individual form of effusion, the fluid and the solid parts are separately thrown out; and, finally, that the further course of the disease depends upon whether and in what wise the substances effused are susceptible of further organic conversion, or whether, on the other hand, their vital energy ends with their formation; and they are no longer subject to any save chemical and mechanical influences. Excepting where the vital powers of the organism are reduced to a very low ebb indeed, a product of inflammation will seldom be completely withdrawn from the control of organic action. The vascular development in the serous membrane strives to make way in even the crude coagulated false membranes; and the collected fluid, though not in immediate contact with an absorbent, vascular surface, may, nevertheless, with the cooperation of endosmosis and exosmosis, undergo divers changes. It is thus, perhaps, that in apparently hopeless cases, accumulations of pus within the pleural cavity have, though rarely, been eventually removed by absorption.

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[1] Villermé, after Dupuytren, in the Dict. des Sciences Méd. Compare, on the other hand, Hodgkin (the Morbid Anatomy of the Serous Membranes, vol. i), Lobstein (Anat. Path. vol. i, p. 236.)

No object in the history of pleurisy has, perhaps, more attracted the attention of pathologists than the *organizing of the inflammatory product*. Various theories have been promulgated touching the mode in which new vessels form in the effused substance. Two, directly opposed to each other, hold the ascendant, and that under high authority.[1] According to the one, the new vessels are immediately derived from those preexisting, through the *vis a tergo* of the heart and arteries, which, by virtue of the undue vascular repletion, and of the increased activity of circulation in the original structure, is directed, partly by mechanical influences, partly by a species of vital attraction, towards the mass about to be organized. According to the other, it is the exudation itself which, by dint of its inherent vitality, causes a movement of molecules,—their transformation into blood-globules, the formation of independant blood-canals, not originally connected with the blood-vessels of the body, so that the circulation in the exuded mass conjoins and becomes subject to that of the rest of the system, only by slow degrees. This latter view has not yet been corroborated by direct observation, and rests rather upon conjecture, deduced from analogy with the first development of circulation within the egg, during the process of incubation, and especially with the mode of extension of the vascular system in the embryo of fishes, as described by Döllinger.[1]

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[1] The idea of the late Everard Home of the vessels originating from minute air-vesicles ranging together in rows, their coalition and final union with the normal vessels of the body for the reception of blood is now, we believe, quite abandoned. It is here alluded to merely because it recalls the notion of fermentation and sanguification being kindred processes.

Here, as usual, theory has gone in advance of established facts; it may not, therefore, be amiss to introduce the original groundwork of the aforesaid hypothesis. Döllinger professes to have observed in the embryo of fishes, a two-fold mode of increase in the minute currents of blood. On the one hand, blood-globules are seen to depart—at first singly, but by and by in augmenting numbers—from the already existing currents, forcing for themselves one or several passages through the animal mucus, so as ultimately to constitute a continuous and permanent stream. Thus far all earlier and living physiologists agree. On the other hand, he frequently noticed in the immediate vicinity of developed blood streamlets, oscillatory movements in the animal mucus; then a moulding of the latter into globules, which appeared gradually to acquire the character of blood discs. Into this oscillatory mass single blood-corpuscles would pass over from the neighbouring blood-current, and then back again; and, after the lapse of about forty-eight hours, a complete new blood-streamlet was to be seen in place of the aforesaid mass. Döllinger remarks, that the oscillation did not appear to him spontaneous, but the result of the *vis a tergo* motion communicated by the neighbouring blood-current. From these data, and from having occasionally observed individual blood-corpuseles to merge in and become incorporated with the mucous mass, Döllinger concludes that blood is animal matter in motion, and animal matter, blood in repose, and upon this inference is founded the hypothesis of an independent development of blood and blood-vessels in the inflammatory exudation. Several later physiologists, however, reject the conclusion. J. Müller (Physiologie, vol. i, third édit.) has not arrived at the same results, nor indeed witnessed similar facts, after numerous researches on the subject. The sum of Döllinger's facts (beyond which we shall not venture to follow him) would amount to this,—that new blood-streamlets are dependent, for their development, upon others previously in existence.

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[1] See Döllinger's masterly treatise on the circulation of the blood (Denkschriften der Königl. Akad. d. Wissenschaften zu München, vol. vii, p. 169. 1821.)

The processes within the egg during incubation, are resorted to in like manner for the purpose of showing, that circulation may establish itself in the products of effusion, independently of the parent structure. The analogy is, however, here too remote to carry much weight. We see, on the one hand, the egg, a production in itself sufficiently complex, with an independent nucleus, out of which every possible variety of organic structure is developed, provided its indwelling, but latent life, is wakened by impregnation, and upheld by incubation. On the other hand, we have, in the exuded substance, an accidental product, differing from excretory matter only in its composition—not identical, but yet homologous with the elementary formative mass. Such adventitious product, by reason of its slender indwelling vitality, can enter into a vegetative, not into a perfect animal union with the organism. We have, therefore, ample reason for discarding an analogy so little in keeping either with real facts or sound logic.

Hitherto, no one has been able to watch the movements of blood-currents in inflammatory products, during life; nor is this species of research feasible, unless through a fortunate concurrence of circumstances. Cadaveric phenomena are, therefore, the more deserving of close investigation. With this intent, I have omitted no opportunity of removing false membranes of every description, either alone or together with the subjacent pleura; and, having first washed them carefully in cold water, proceeded at once to spread them on glass plates to dry. As the congested blood-canals in inflamed or newly-organized textures, do not part with their contents to the larger veins, as in healthy parts, I have been able to furnish a series of preparations in which the course of at least the larger of the newly-formed branches is conspicuously shown by a surprisingly delicate distribution of blood-vessels.[1]

Wherever vessels had formed in the adventitious membranes, they proved to be continuations of the branches ramifying in the serous coat; they penetrated the false membrane at numerous points, and then branched out in a stellate manner, or formed into partly divergent, partly parallel, fascicular groups. Hodgkin describes them similarly, (l. c. p. 51.) Such was the character of the vascular development, more particularly in the gelatinous form of exudation, which, in this respect, so nearly approaches the normal formative substance, as, after a short period, to display ramifications of vessels precisely similar to the parent ones, and, in all probability, created in the manner witnessed by Döllinger in the embryo of fishes. I have repeatedly and very carefully taken up, upon pieces of glass, detached and floating fragments of the gelatinous product in question, and searched for independent vascular development, but in vain; and I feel convinced, that if such flakes should ever be found to contain blood-vessels, they must originally have clung to the pleura, and been severed subsequently.

These processes do not take place quite so readily in the less organizable false membranes. Here the descriptions given by Laennec and Gendrin are especially applicable.[2] On the serous membrane appear a multitude of little red warts, like clusters of protruding vessels: these insert themselves into corresponding pits in the false membrane, upon the surface of which arise little arborescent or stellate extravasations of blood. By degrees these blood-red ramifications, which are rude in shape and surrounded by imbibition, so as to seem much thicker than the blood-current itself, become more strongly marked. "These vessels," says Laennec, "present an outer and softer layer, formed by desiccated blood; which layer incloses a small whitish cord of coagulated fibrin, apparently hollow in the centre, and pervious to the blood-stream." These shapeless, thick-walled canals, gradually change into the natural coating of a vessel, which, pursuing its course in the same manner, thus seeks to traverse all the organizable portions of the false membrane.

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[1] This is the more advantageous, because the artificial injection of inflamed structures is confessedly very imperfect, and, where a high degree of inflammation is present, impracticable.

[2] Laennec (Traité de l'Auscultation médiate, vol. i, p. 337 :) compare Dr. Forbes's translation, 3d ed. Gendrin (l. c. vol. ii.)

Some plastic substances are possessed of so little power of attraction over the capillary vessels, that neither those little vascular acumina form upon the pleura, nor the little dotted or stellated extravasations upon the new membrane. Such heterogeneous formations go on irritating the pleura, until a third membrane is thrown out, which, acting as an intermediate vascular link, establishes something approaching to vegetative reciprocity between the two, and being itself devoid of nerves, obviates morbid irritation.

Whatever does not sooner or later yield to the assimilative power of the vessels, and consequently is neither converted into vascular cellular tissue, nor removed by absorption, becomes perfectly isolated and encysted, and may be thus retained for years, without proving fatal, or even occasioning any very urgent symptoms.[1] Thus, after all the fluid parts have vanished, the solid constituent of the effusion frequently remains behind, a pap-like mass of the consistency and aspect of moist cheese, or of imperfectly coagulated white of egg. Previously to this, the effused product usually sinks to the most dependent portion of the cavity of the pleura, so that the above mass accumulates into a layer more or less thick, between the two firmly adhering and thickened pleuræ, posterior to the inferior lobe of the lung. When tubercle enters into the combination, and does not prove rapidly fatal, either through its destructive tendency as a local irritant, or through the constitutional affection whence it arises, it is occasionally met with, as a residue of the aggregate morbid product, in small scattered portions included betwixt the pleuritic adhesions. Those isolated masses are, however, susceptible of further transformations, and it is highly interesting to mark the various expedients that Nature adopts for abating their prejudicial tendency on surrounding textures.

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[1] A very remarkable instance of extensive empyema persisting for four years under variable symptoms, is related by B. Mohr (Beyträge zu einer Monographie des Empyems, 1839—case 15).

Most frequently, a deposition of phosphate of lime takes place little by little, until the whole is converted into a hard earthy concrement, constituting an irregular, rough plate, sometimes separable into two layers, which inclose an intermediate residue of the caseous, atheroma-like mass already alluded to. The plates in question are wont to adhere so firmly both to the parietes of the thorax and to the surface of the lungs as to prevent the two folds of pleura being distinctly recognized. These relations apply to most instances of so-termed ossifications of the pleura, which would seem, even where they form in the pleura itself, or in the cellular tissue external to it, to originate in inflammatory action (Hodgkin).[1] The thin lamellæ occasionally found without any accompaniment of pleuritic adhesions, and the fibro-osseous tumours of the pleura, are obviously referrible to a different source.

Sometimes the fluid is removed, not by absorption, but by the shorter process of forcing a passage out of the body. This occurs most readily through the pulmonary substance; and it is remarkable, that the lung is perforated, chiefly in its superior anterior portion, not low down; in the majority of cases, at the inferior surface of the upper and middle lobes;—not where the lung has suffered compression, but where it has continued to expand. Such portions are commonly attached by old adhesions to the costal pleura, and form an arch over the effusion, whose progress within the pleural sac is thus arrested. The fluid not being elastic, presses against this arch, until at length some point of the pulmonary substance softens, gives way, and an escape is effected through the bronchia. Where the perforation occurs at the inferior lobe, or at the base of the lung, as is the case now and then, that part has been protected from compression by old adhesions, and has remained partially pervious to air. These perforations are either oblong or rounded,—rarely exceeding two or three lines in diameter, and smooth at their edges. There is commonly but one; Andral, however, once encountered two in different spots. The parenchyma in the vicinity of these pulmonary fistulæ, is, within the extent of about half an inch, in a state of gray hepatization, or else of complete purulent softening. The accident generally proves fatal; a few examples of recovery are, however, adduced by Andral (Clin. Méd. vol. iv, p. 561;) and by Heyfelder (Studien, vol. i, p. 26). Gendrin relates an instance of the fluid forcing its way into the anterior mediastinum.

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[1] Compare the chapter on Pericarditis. A most remarkable case is related by C. H. W. Posselt (De pleuræ ossificatione, Heidelberg, 1839), which might serve as an illustration of pleurisy in all its phases. Posselt has collected a considerable number of examples of ossification of the pleura. He found that in 27 cases the right side was affected in 12, the left in 15; and that, out of 34 cases, 30 applied to men, and only 4 to women; which numerical relations favour the notion that these ossifications are of inflammatory origin.

Escape of the effusion through the thoracic parietes (*empyema necessitatis,*) holds out a better chance for the patient. Here, recovery has been known to take place very rapidly; in other cases, again, to require years for its completion. The perforation never occurs at the base of the thorax, but generally between the third and fourth, or between the fourth and fifth ribs,[1] and either at the side or in front of the thorax. There is, first, œdema of the external integuments, which is however presently converted into a vast fluctuating abscess. Where the external skin or the aponeurosis offers too forcible resistance, the pus diffuses itself through the subcutaneous cellular texture, and between the muscles, producing extensive infiltration, fistulous channels, caries of the ribs, and finally death, with hectic symptoms. Where the matter discharges itself between the ribs, the lung is mostly free from adhesions and thoroughly compressed. In such instances, recovery depends upon the capability of the lung for speedy and complete re-expansion, a contingency incompatible with the presence of very thick layers of plastic exudation, or with a very firm, perhaps cartilaginous condition of the investing false membrane. Andral often failed, by inflation after death, to restore a lung so circumstanced to its natural volume. Heyfelder and Hodgkin are therefore perfectly right in impugning the success of paracentesis of the thorax, unless performed at an early period.

One reason why spontaneous evacuation through the bronchia and thoracic parietes often proves fatal, is the noxious influence exerted upon several forms of effusion, by the air penetrating from without. In most instances the air very soon becomes decomposed, assuming a character of indescribable fœtor. The consequence is, not only abiding irritation of the pleura, but likewise, in the instance of bronchial fistula, fatal inflammation of the substance of the lung, or at least of the bronchial and tracheal mucous membrane. Only certain effused substances, however, suffer decomposition of this nature. Heyfelder never once met with it (p. 304); Davy (Phil. Trans. 1823) examined the gas, present in pneumothorax, and found it to consist of 0·92 nitrogen, and 0·08 carbonic acid. The fluid of empyema when brought in contact with atmospheric air, generally evolves the odour of sulphuretted and phosphuretted hydrogen.

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[1] Heyfelder once saw the rupture occur between the sixth and seventh ribs. (See his valuable treatise on Chronic Pleurisy, 'Studien im Gebiete der Heilwissenschaft, vol. i, p. 39.)

The passage of empyema through the diaphragm is rarest of all. Andral (Clin. Méd., vol. iv, p. 471) relates one instance: the perforation was an inch and a half in diameter, its edges uniform and smooth, the contiguous muscular tissue appearing healthy; the peritoneum was forced down; the effusion into the cavity of the belly was bounded by adhesions, and by a thick, flocculent adventitious membrane, between the spleen and the abdominal walls. Mohr (l. c. p. 148) adduces two cases, the one perfectly corresponding with Andral's,—the other presenting a perforation of the diaphragm near the vertebral column, and the descent of the pus behind the peritoneum, along the psoas muscle, with abscesses and fistulous channels in the thigh, extending down to the knee.

The pleural cavity being surcharged with effusion, not only the thoracic, but likewise the proximate abdominal organs are of necessity both mechanically and dynamically oppressed; and this in a degree proportionate to the duration of the pleurisy to the heterogeneous character of its products, and to the more or less protracted sojourn of the latter. The lung of the side affected suffers most; its compression being commensurate with the increase and ascent of the fluid, until it is no longer capable of expansion, and air can scarcely enter the simultaneously compressed bronchial tubes. If the lung be free from previous adhesions, it is pressed upwards and forwards; and, finally, on all sides towards its root, where the bronchi and blood-vessels penetrate. It now occupies the least possible room, and might in the shrivelled and flattened state in which it lies, in front of the vertebral column, be looked upon as utterly decayed and evanescent. The different lobes are mostly adherent to each other,—seldom freely and separately suspended in the fluid. The parenchyma is inelastic, devoid of crepitation, bloodless, and, for the most part, quite passive; for where tubercular disease, for instance, develops itself, or advances, during the progress of chronic pleurisy, a thing by no means rare, compressed portions are almost always found exempt from that disease.

About this period, the bronchial branches are usually loaded with a tough whitish mucus. Where, on the contrary, firm adhesions of an earlier date are present, they retain portions of the lung expanded, and these always remain at least partially pervious to air, in spite of the tendency of the effused fluid to intervene on all sides. In this way pleuritic fluid is often found pent up between such adhesions.[1] It is astonishing with what rapidity the lung again expands, and resumes its normal function, after absorption of the fluid; it is only when encumbered by very tough false membranes; or when its elasticity has become impaired by protracted sojourn of the effusion, that it continues for a longer period, perhaps for life, in a state of collapse. This of course applies with greater force to the inferior lobes.

On the healthy side the lung dilates, as Laennec first showed, vicariously, and in a degree corresponding to the increased call upon its function. Its expansion could only be checked by the amount of effusion being sufficient to impel the mediastinum against it; a case which does not appear to have occurred.

In left-sided pleurisy the heart is frequently driven from its natural position. Its pulsation is then more distinct towards the sternum, and mostly somewhat higher up than usual; sometimes, however, though less frequently, lower down, towards the epigastrium. In the right cavity of the pleura, effusion must be very considerable indeed, to force the heart either farther to the left, or downwards. A subsidence of the heart towards the vertebral column is likewise rare. The organ gradually assumes its normal position, in proportion as the fluid is removed by absorption.[1]

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[1] The mode in which compression of the lung is brought about, where adhesions are present, has been very minutely investigated by Mohr (l. c. p. 127, et seq.) Out of 43 cases (6 of which were double), partly of his own observation, partly compiled from the works of Andral, Laennec, and Louis, the compression and displacement of the lung occurred.

Once in the direction from above, downwards.
4 times from behind, forwards.
4 times from before, backwards.
4 times from within, outwards.
13 times from below, upwards.
23 times from without, inwards.

After seeking to obtain room by the displacement and compression of the thoracic organs, the morbid product, by depressing the diaphragm, distending and thrusting forward the intercostal muscles, and elevating and expanding the lower ribs, effects at length a positive dilatation of the affected side. A fulness and convexity are thus given to the corresponding hypochondrium, the liver and spleen, respectively, projecting from beneath the margin of the false ribs; which is more apparent when the patient is sitting up than when recumbent. The involved side is now found, on measurement, considerably to exceed the healthy one in circumference—sometimes to the extent of a couple of inches.

Stokes (Dublin Journ. vol. ix, No. 25, 1836) first pointed out that the above dilatation is not simply of mechanical, but rather of dynamical origin, being dependent upon a paralysis of the diaphragm and intercostal muscles, the effect of neighbouring inflammation. He never observed the vaulting of the hypochondrium, or the distension of the intercostal spaces to take place at the commencement of pleurisy, although the effusion might, even then, be very considerable; nor in fact until the disease had lasted some time, and the innervation of those muscles been impaired. Under the latter circumstances he sometimes found the diaphragm to give way almost suddenly, and directing its convexity towards the abdomen, force down the liver, without there being any reason to suspect coincident increase of the effusion.[2] Laennec, on the other hand, sometimes noticed a dilatation of the affected side to occur within a few days of pleurisy setting in (l. c. vol. ii); it should, however, be stated that this early dilatation is very inconsiderable, if not altogether a delusion, arising from the affected side not participating in the movements of respiration.

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[1] See H. Clarus (De cordis ectopiâ, Lips. 1839), who gives a minute account of this condition.

[2] In proof that the dynamic explanation of this symptom is well founded, Stokes mentions that, neither in symptomatic hydrothorax, in emphysema of the lung, nor in hypertrophy of the liver (nowhere, in short, except where the muscular fibres were paralysed by inflammation) has he found the intercostal spaces bulging forward

In chronic pleurisy, no sooner is the effused matter absorbed, than the parietes of the thorax collapse, and, as shown by Laënnec, a contraction ensues, which lasts for a longer or shorter period, perhaps during life. That side of the chest becomes flattened, the ribs maintaining the same position as during the most forcible expiration; the shoulder and nipple are both lower, and there is frequently a slight incurvation of the spine towards the affected side; according to comparative admeasurements of both sides of the thorax, the contraction sometimes amounts to a couple of inches (Mohr's twenty-first case) in the transverse, and to more in the longitudinal diameter. This condition is observable, as well after absorption, as after escape of the pleuritic effusion, through a spontaneous or artificial opening in the chest. In some instances, the very firm false membranes surrounding the lung on all sides, prevent its perfect re-expansion; or the substance of the lung may, through the protracted sojourn of an heterogeneous product, become so impaired in elasticity, as to be incapable of immediately resuming the exercise of its function. A more influential cause is, however, in all probability, the paralysis of the diaphragm and intercostal muscles assigned by Stokes. For those muscles remaining inert during the process of absorption, the lung cannot inhale sufficient, if any air, and atmospheric pressure will consequently compel the wall of the thorax to fill up the void caused by removal of the pleuritic effusion. The more intensely the muscles are affected, the longer will their paralysis endure, and the more striking, during that period, be the deformity of the trunk, confirming Stromeyer's theory, as to the origin of lateral curvature of the spine. Upon the whole, this lateral contraction is fraught with less general disturbance than might at first sight be apprehended.

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and distended, but, on the contrary, always distinctly marked and tense, although, in those diseases, the hypochondriac region is wont to protrude considerably. I can confirm the accuracy of this statement.

By far the most ordinary sequel of pleurisy are adhesions between the lung and the costal pleura, and between the lobes. Two different forms may be distinguished: first, the cellular, in which the two surfaces of the pulmonary and of the costal pleura are, within a smaller or greater compass, equably united to each other through a dense, astricted cellular tissue; and, secondly, the filamentous or band-like, wherein the two sheets of the pleura are bound together by separate bridles, having a smooth exterior, closely resembling that of the serous fabric. These intermediate structures appear identical in all respects with the serous membrane, whose product they are, and into which they immediately pass. They consist, in like manner, of densely-stratified cellular tissue, with an investment of epithelium-cells; they are often supplied with largish blood-vessels, and sometimes have fat within their texture, (Laennec.) This I have frequently witnessed. These adhesions may persist during life without occasioning any sign of disease, or may disappear in course of time. In the latter instance, the membranaceous cords lengthen, become thinner about their middle, and finally rupture; both ends then curl up, and nothing except a white, scar-like thickening of the serous membrane remains. Opportunities for tracing this process upon the serous membranes are not rare. The most illustrative cases are those recorded by Béclard aud Dupuytren. (See Villermé in the Dict. des Sciences Médicales.)

The adhesions met with in tubercular disease of the lungs, differ from those of genuine pleurisy. They commence at the apex, and gradually descend to the base of the pleural cavity, closely following the course of the tubercular development. They are less the result of decided inflammation, than of a chronic irritation, limited in degree, and kept up by the proximity of the heterologous product. The result is complete blending of the pulmonary with the costal pleura, accompanied by lardaceous thickening and degeneration of the serous structure. Through this intimate fusion is achieved the passage of the intercostal and other vessels into the diseased substance of the lung. (See Tuberculosis.) Under certain conditions, this slight irritation, calculated only to promote insensibly the adhesion of patches of the pleura immediately at hand, may amount to true inflammation, which then rapidly overspreads the pleura, throwing out diverse products, for the most part connected with tubercle, and usually passing into a chronic pleurisy, which terminates only with life. The complication of pleurisy in tubercular disease of the lungs is so common, that Mohr encountered it in 15 cases out of 20.

The diffusion of inflammation along the serous surface is variable, and for this reason several distinct forms of pleurisy are admitted. It is rare that both sacs are simultaneously inflamed: out of 35 fatal cases which came under my notice, only 9 were double pleurisies, and in 5 out of the 9, one sac was implicated only in a minor degree. Both sides appear to be almost equally prone to the disease, with this difference, that pleurisy of the left side is both more likely to prove fatal in the acute stage, (in those 35 cases the left side was the seat 16, the right, 10 times,) and more apt to pass into a chronic state. (In 56 cases adduced by Mohr, the left side was 37 times the seat, the right 19 times.)

The disease is often limited to particular patches of pleura. For this restriction various causes may be assigned, the general tendency of inflammation in serous membranes being to spread superficially. It may be owing to some specific morbid cause, as tubercle, fungous growths forming beneath the pulmonary pleura, or to phlebitic lobular abscesses in the lungs. Or, again, the inflammation may have been originally of an adhesive character, promptly occasioning adhesions of the pleura, not redissolved on the disease afterwards assuming a more violent character, and therefore serving to arrest the diffusion of the morbid product. It is, however, mostly old adhesions resulting from preceding attacks, that stay the progress of the inflammation, and give occasion to partial pleurisy and circumscribed empyema. Andral distinguishes five forms of partial pleurisy, according as the effusion is restricted to circumscribed patches: first, between the pleura and the lung, (circumscribed costo-pulmonal pleurisy;) secondly, between the lung and the anterior, or posterior mediastinum, (circumscribed pleurisy of the anterior or posterior mediastinum;) thirdly, between the base of the lung and the diaphragm, (circumscribed diaphragmatic pleurisy,) which is wont to assume a very peculiar and perilous character; and, finally, in the interlobular spaces, (circumscribed interlobular pleurisy), a very rare form.

However frequently the pulmonary pleura may participate in inflammation of the substance of the lung, the converse seldom happens. The effusion within the cavity of the pleura would appear to compress the lung, ere its parenchyma has had time to share in the inflammation. On the other hand, the neighbouring peritoneum, and especially those portions of it which invest the liver and the spleen, are in most instances implicated from the very first, although in a minor degree, so as only to produce adhesions. Where, however, the pleuritic product has the tubercular character, the latter is equally cognizable upon the peritoneum itself, and in its effused product. The pericardium is, perhaps, rather less frequently involved. Sometimes it contains a vast amount of limpid fluid; sometimes, fluid rendered turbid by the intermingling of delicate flocculi; sometimes, again, there is gelatinous effusion with partial adhesion; or, lastly, a still higher grade of inflammation. Within the heart's cavities, especially the right, a large proportion of fibrinous coagulum invariably occurs.

Rheumatism, which evinces a marked predilection for the serous and fibrous structures, is unquestionably the most ordinary source of pleurisy.—It has been already shown that the passage of pus into the circulation, especially in phlebitis, (see the respective chapter,) sometimes leads to a general or circumscribed inflammation of the pleura. The development of tubercle or of fungous growths, gangrene of the lung involving the pleura, and the discharge of softened tubercle into the thoracic cavity, all give rise to pleurisy. Amongst the traumatic causes, violent contusions, penetrating wounds of the chest, and fracture of the ribs, are the most common. The influence of sex is remarkable. In females the disease is far less frequent than in males, and its transition into a chronic state particularly rare in the former sex, in whom it is almost entirely restricted to those of a tubercular habit. Pleurisy is not confined to any particular period of life, although less frequent in infants and in very advanced age. The majority of cases occur between the fifteenth[1] and the fiftieth years. I purposely abstain from any numerical statement, because the experience of other writers, like my own, is principally derived from hospitals, at which children with acute diseases are seldom received, and females in a less proportion than males.

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[1] Valleix (Clin. des Enfans, nouveau-nés. 1838. pp. 69, 198.)

# CHAPTER II.

## INFLAMMATORY DISEASE OF THE SUBSTANCE OF THE LUNG.

### SECT. I.—PNEUMONIA.

It was long before the anatomical characters of inflamed substance of the lung became accurately known, or suitable means indicated for discriminating between true pneumonia and congestion, softening, discoloration, and impermeability of the pulmonary cells, whether separate or combined. Pneumonia may well be said to have tended more than any other subject of pathological research to illustrate the doctrine of inflammation generally. In spite, however, of our extended knowledge of the disease, certain points still remain obscure; amongst these may be enumerated the pneumonia of infants, and that termed hypostatic. Whatever either the investigations of myself or others may have elucidated with respect to those debateable points, shall be duly set forth after describing ordinary pneumonia as it occurs in the adult.

The course of pneumonia is characterized by several stages, no less plainly depicted during life than after death. On opening the thorax in the first stage of pneumonia, we find the lung more than usually distended; it does not collapse, as usual, from the pressure of the atmosphere, but retains its volume, as if inflated. It is heavier than a healthy lung. Its surface is of a brown-red, alternately lighter and darker. Morgagni, together with other pathologists, mentions the occasional occurrence of a deeper blueish black tinge. This brown-red, or blue-red marbling is, however, not well defined, but irregularly blended, both the transitions and the colours themselves presenting a dingy aspect; the whole lung is wanting in smooth serous lustre; it has a dull look, the more the inflammation approaches the surface, and the more the pleura is affected. The lung pits upon pressure like an œdematous limb, and is far less tough and elastic than when healthy; its texture is soft and friable, the natural crepitation is considerably diminished, and only here and there perceptible. On incision, a large quantity of frothy, and more or less deeply tinged sanguineous fluid escapes. The cellular structure is indistinct. The mucous membrane of the finer bronchial ramifications is of a brownish red, and covered with a tenacious rust-coloured fluid. Gendrin first discovered that a lung, thus circumstanced, does not recover its natural colour and condition by repeated washing and maceration in water. Aqueous injection decolours the confines only and not the inflamed portion. Hereby is inflammation distinguished from mere congestion, infiltration, and sanguineous hypostasis.

Gendrin states that when coloured fluids thrown into the pulmonary artery penetrate to the capillaries of the pulmonary cells, and from thence into the cells themselves, and the minutest bronchial twigs, there is no inflammation present. By these injecting fluids not filling the minutest ramifications, or at least not the vascular network of the cells, it is ascertained that the inflammation proceeds from the interstitial texture,[1] and not from the aerial cells. With a view to ascertain the peculiar characters of pneumonia as originating in the air-cells, it has been attempted to excite, in animals, artificial inflammation of the lungs, by introducing corrosive gases and other irritant substances into the air-passages. Cadaveric inspection has since made us acquainted with these relations in man. The lung then appears replete with millet-seed shaped granules, which are by no means to be confounded with tubercles. These granules consist of a very tough, yellowish-white mass, giving to the surface of the inflamed part a punctate appearance of a more or less pink colour. Gendrin maintains, that in the other case, namely, when the inflammation proceeds from the interstitial texture, the more delicate bronchial twigs retain their natural light colour; I have, however, seen them sometimes of a reddish-brown; sometimes of the natural colour, so that the aspect of the subjacent tissue shone through. All these differences are not very obvious during the first and second stages, and cease to be discernible after the pneumonia has reached the third stage. Gendrin's method of proof is attended with difficulties, for the injection of inflamed lungs never completely succeeds, the inflamed portions taking up the mass at their circumference only, and never where an inflammatory product is once established. But these experiments, to which so much importance has been attached, are inconclusive, not only because inflamed textures evince no permeability of their minutest vessels, but likewise because a delicate injection, owing to the multiplicity of anastomoses, is incapable of distinctly marking the boundary between the system of the pulmonary and the system of the bronchial vessels; wherefore the minute bronchial twigs may possibly become simultaneously injected. Practically speaking, it is of subordinate interest to determine the exact *point de départ* of pneumonia; it is sufficient to know that inflammation of the parenchyma of the lungs may either originate within the capillary system of the organ, or be derived from the mucous membrane of the air-passages, or what is least common from the pleura.

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[1] Pneumonia interstitialis et vesicularis. Hourmann and Dechambre.

The *first grade* of pneumonia seems to consist, essentially, in the capillary vessels of the parenchyma being gorged with blood, whereby the pulmonary cells, ordinarily filled with air, become, for the most part contracted, and more solid than before. The blood contained within the capillaries, has also undergone a change, both in a physical and in a qualitative point of view. For, even in this stage, there appears to occur, in most of the capillaries, a real stagnation, a stand-still of the blood-corpuscles,—causing their colouring matter to dissolve, and to become intimately blended with the serous portions of the blood. The walls of these little vessels are now so far changed as to admit of an exosmosis of their contents, and the tenacious, rust-coloured, and semi-transparent sputa form in the pulmonary vesicles, and the minutest bronchial tubes.[1]

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[1] Gluge (Patholog. mikroscop. Untersuchungen, fasc. i, p. 58,) says of the first grade of pneumonia: "In the capillary system of the lungs the following change occurs: the blood-globules collect into the composite spheres described, and these unite through the medium of a white fibrinous mass. One drop of the fluid accumulated in such a lung contains hundreds of them, and the parenchyma of the lung is equally replete with them," &c.

Viewed under the microscope these latter display, within an amorphous, or slightly granulated mass, a tolerable quantity of blood-discs, a proof that, at the outset of the inflammatory stagnation, the smallest vessels undergo partial rupture. This first stage is designated by the term *sanguineous infiltration*, splenization (*Engouement*.)

With the advance of inflammation, the *second stage* is established in the midst of the involved part. This change is often very rapid,—two or three days sufficing,—more especially in individuals young and vigorous, or otherwise prone to plastic exudation. In rarer instances, the first stage is of longer duration, lasting for ten days or upwards, and then passing, if there be any tendency to the formation of heterologous products, into a chronic state, or proceeding, promptly to the third and commonly fatal stage. Thus, here, as in inflammation generally, the period of effusion decides the ulterior course and final issue of the disease. In this stage the lung is dilated to its utmost capacity, its specific gravity is augmented, (being, according to Gendrin, when compared with the other lung, as 1·15 or 1·19 to 1.) condensed throughout, without a vestige of crepitation, of a lightish brown-red colour, and greatly softened, so as to be pretty easily broken down with the finger, or crushed between the finger and thumb. The cut surface appears uniformly studded with flat granular elevations, which are easily effaced. These granulations appear to result from the filling of the air-vesicles with effused matter, as may be inferred from their variable size; for in children they are hardly appreciable (the section being almost smooth); whilst in old persons and in emphysematous lungs, they are of magnitude corresponding.[1] Of the real lung, nothing is observable, except the larger bronchi and the blood-vessels,—all the rest forming one homogeneous mass. In older persons the surface of a section, owing to the larger proportion of black pigment, acquires a granite-like aspect. In cachetic individuals, the colour is a dirty mixture of gray and green, with a shade of red. When old tubercles are present, they are found completely softened and liquefied in the midst of the inflamed texture. In proportion as the subject was young and vigorous, the disease of short standing, and the second stage little advanced, the colour approaches more nearly to a brown red. The cut surface is rather dry; on, however, compressing or scraping the part, a little frothless fluid, mostly of a dirty pale red, or faint brown colour, and sometimes resembling wine lees, escapes.

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[1] In an instance of considerable emphysematous dilatation, in a man who died of pneumonia, the diseased patches of the lung presented such peculiarity of appearance, that I at first entertained doubts respecting the real nature of the malady. The cut surface appeared as if besprinkled with dull-yellow granules of irregular shape, mostly of the size of hemp seed, and of the consistency of soft butter or thick pellets of pus. These granules imperfectly filled little smooth cavities, likewise of irregular shape. They might have been mistaken for tubercles: all doubt, however, was removed by their aspect, their seat in pulmonary cells obviously dilated, their gradual transition into the ordinary bronchial mucus, their occurring at the anterior portion and at the margins of the inferior lobe of the right lung, and in the midst of gray hepatization (presenting in a conspicuous manner all the gradations from undilated to thoroughly

In this stage, the gorged capillaries have thrown out their soft contents (decoloured blood, serum, and fibrin,) into all the interspaces. Hereby the textures have lost their proper character, and become uniformly macerated. The decomposed blood within the pulmonary cells is now transformed into a coagulated mass (plastic lymph) of very slender consistency.[1]

Viewed under the *microscope*, the exudation of genuine pneumonia reveals a distinctly granulated condition, and, generally speaking, an elementary composition varying according as this second stage is more or less advanced. Numerous examinations have induced me to believe that in this kind of pneumonia, the effused substances are originally fluid; then coagulate to a tolerable degree of solidity; and finally again liquefy. In the primary form, those substances, as above stated, display a number of blood discs, imbedded in a nearly amorphous, slightly granulated, or striated mass. On being treated with water, this mass still exhibits no cells (beyond a few shed ciliary cylinders), but numerous elementary granules, either scattered singly, or collected in groups. Acetic acid dissolves the greater portion of the mass, leaving unchanged only the elementary granules,

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emphysematous parts), and finally, by the fact, that no tubercles were discoverable elsewhere; to which may be added, the symptoms observed during life.

[1] Gluge (l. c.) says: "Even under the microscope the hepatized mass no longer exhibits any trace of air-vesicles. The pulmonary cells are filled with fibrin, and the whole is converted into a uniform substance, wherein can be discovered, after removing the effused matter, the spiral pulmonary fibres, and an occasional blood-vessel with consolidated blood."

and more or fewer spherical nuclei of various magnitude. When some time elapses before the coagulation of the effused substances takes place, these nuclei become previously sheathed in spherical cells,—*exudation-cells.* After coagulation, the mass becomes amorpho-granular; by mechanical division, a display of certain forms only—for the most part exudation-cells—is sometimes produced; by treatment with acetic acid, this mass, again, is almost entirely dissolved; those elementary granules and spheroid nuclei alone remaining. With the ultimate liquefaction of the coagulate effusion, the *true* development of cells appears to take place; and it now depends upon relations not clearly definable, whether more exudation-cells or more pus-globules are developed. These two last forms constitute by far the greatest portion of the mass, and become readily discernible, on the application of acetic acid. In this variety of pneumonia, granule-cells appear to form in very small number, and often not at all.[1]

This condition is termed *hepatization,—red softening.* During this period, the disturbance in the respiratory function has attained its highest pitch, unsusceptible of augmentation, even during the ensuing stage. Although at the very commencement of the disease the difficulty of breathing is manifested by the distress of the patient in a very troubled and striking manner, it is not until after an extensive portion of the lung has become thoroughly hepatized, that the intense constitutional signs of impeded respiration and impaired sanguification are conspicuous. For the blood, prevented from circulating freely through a portion of the ramifications of the pulmonary artery, and thus withheld from free contact with the atmospheric air, is only partly arterialized, and therefore passes into the left ventricle of the heart, and into the general circulation, in a predominant venous condition. On its reaching the encephalon, the function of the brain is disturbed, and in many instances coma, and a state resembling that which results from poisoning with carbonic-oxide gas, set in. The change in the crasis of the blood is likewise portrayed in the altered complexion, which, after hepatization, usually acquires a sallow hue. This sometimes almost icteric tint may perhaps now and then arise from the stagnation of venous blood in the liver, which is frequent in pneumonia, but is by no means always referrible (as older authors, and Stoll in particular maintained) to that organ taking part in the inflammation. I have seen it repeatedly in hepatization, not only of the right, but likewise of the left lung, and even in cases in which, after death, the liver was found in quite a healthy state.

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[1] These microscopic relations would hardly justify the adoption of the epithet "croupal," as applied by Rokitansky to genuine pneumonia, inasmuch as the false membrane in real croup displays a different elementary composition.

The change of position of the individual lobes, in hepatization, is of great importance in tracing the progress of pneumonia by percussion and auscultation. The inferior, the most frequently affected lobe, enlarges posteriorly, its apex being often elevated to above the third rib, whilst in front it is apparently of the breadth of a couple of fingers only. The middle lobe, and the superior half of the upper lobe, occupy almost the whole anterior surface of the thorax, whilst the inferior, and half of the superior lobes, cover, in equal proportions, its lateral surface.

In the *third*, or final stage, the lung remains the same in respect to volume, density, and total absence of crepitation; but the softening is more advanced, so that by merely seizing a portion of the lung between the finger and thumb, it may be squeezed to a pulp. As a proof, however, that the suppurative process is restricted to the inflammatory product thrown out into the pulmonary vesicles, the cellular structure of involved portions of lung may be restored by careful ablution. The third stage is characterized by a dirty-light gray, or a pale straw-yellow, which shades are modified according to the age and constitution of the individual. A section of a lung in this state still appears granular; the granules are, however, more irregular, and flatter, and they liquefy when exposed to the air. The purulent fluid is for the most part uniformly infiltrated throughout the whole pulmonary texture, in rare instances choking up the air-passages; or it occupies certain portions only, in the shape of abscesses. These latter are frequently created artificially by the pressure of the fingers while removing the lung. Wherever an incision is made, yellowish-gray fluid exudes in considerable quantity, and a few scattered granules remain in the texture; these are somewhat consistent, of a gray-yellow, or rather of a reddish tone, and must not be confounded with tubercles.

Within the infiltrated spot, the mucous membrane of the bronchia looks paler, and of a light violet colour.

It is evident that, in this stage, the sero-sanguineous effusion poured into the pulmonary cells, and there coagulated, is converted into a mixed fluid holding suspended a number of real pus-globules, whereby the utmost degree of softening is communicated to the tissues.[1]

This, then, is the stage of *suppuration*, (purulent infiltration, gray softening, gray hepatization.) Suppuration begins at one or several points of a hepatized portion of lung; nay, each individual air-cell must be considered as a separate sphere of suppuration, so that the coagulate fibrin partly liquefies, partly changes into free exudation- and pus-corpuscles.[2] The suppuration, for the most part, spreads very rapidly, without however time being allowed for its taking up so much room as the fibrinous exudation, death commonly ensuing shortly after the commencement of the third stage. The dirty gray appearance of the suppurating portion of lung, arises from the admixture of the purulent fluid with black pigment, and therefore deepens more and more with advancing age, whilst in young individuals it nearly approaches to the ordinary yellowish tinge of pus. In the course of the third stage, the circulation through the diseased portion of the lung appears almost wholly interrupted; at least the smaller twigs of the pulmonary artery, and sometimes also of the pulmonary veins, are found replete either with clotted blood or with fibrinous concrements. They have sometimes been found to contain redissolved fibrin commingled with pus-globules, just as, in gray hepatization, pus has been detected with the microscope, in blood taken from the heart. It is highly probable that the adynamic phenomena, common during this period of pneumonia, are in a great measure the effect of this contamination of the sanguineous mass. In some instances the examination of other organs, after death, amply verifies this. I once found impacted at the apex of the left ventricle of the heart, irregularly spherical fibrinous masses, liquefied at their centre; and I have repeatedly met with the wedge-shaped products of effusion, alluded to in another chapter, in the tumefied and pale brown-red spleen.

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[1] "However various may be the opinions concerning the nature of gray hepatization, repeated microscopic research has convinced me of its being a true suppuration of the lungs, in general scattered over many points, and in that case not easily recognized with the naked eye." (Gluge, l. c. p. 60.)

[2] There appears to be, between pus and fibrin, a great chemical affinity; for, in the first place, the latter is separated from the blood by the former (see Phlebitis); and secondly, coagulated fibrin is, by contact with pus, always resolved into a pale-yellow viscid fluid.

In all other organs we meet, during pneumonia, with the signs of stagnation, and in some measure of decomposition of the blood. The bronchial glands are swollen and completely softened, their texture of a brown red or light gray, interspersed in aged persons with black pigment. The veins of the great circulation, especially those of the head and neck, are gorged with blood. Small, dark ecchymoses are often seen in the conjunctiva. Beneath the arachnoid membrane is accumulated a considerable quantity of serous fluid; and within the heart, on both sides, masses of black coagulated blood and extensive fibrinous concrements. The liver is highly congested, swollen, and of moderate consistency,—dark coloured, where the disease has proved fatal at an early period,—pale, and wanting in elasticity, where it has been protracted. The spleen is of a deep purple, and very soft; the muscles dark-red, presenting considerable, but not long abiding cadaveric rigidity.

The pleura is mostly implicated in inflammation of the substance of the lung, constituting a true pleuro-pneumonia. The effusion is, however, seldom copious, amounting only to a few ounces of slightly turbid or purulent fluid, with very slender false membranes. Pneumonia is not, as some contend, necessarily conjoined with affection of the pleura; instances occur (see Andral Clin. Méd. vol. iii, p. 372, fourth Ed.) in which no vestige of change was visible in that membrane, so that the scanty portion of dirty reddish fluid discovered, must have been the product of cadaveric exosmosis.

Pneumonia spreads in its course, from below upwards, from behind forwards, and from right to left: this, however, varies according to the form of the disease. Antecedent morbid changes in the pulmonary substance, may modify its diffusion in various ways. Thus, in a case where, owing to by-gone pleuritic effusion, the base of the right lung had not recovered its pristine elasticity,—having, perhaps, become partially obliterated,—I found the whole lung in a state of red hepatization, with the exception of the apex, the anterior margin, and the base, which latter is not wont, under other circumstances, to remain exempt. Ordinary pneumonia mostly invades an entire lobe of a lung, or at least the greater portion of a lobe, before it passes into the neighbouring one. (Lobar pneumonia.) It is rare to find the lowest portion of the inferior lobe, and at the same time the corresponding portion of the uppermost lobe hepatized, leaving the upper half of the inferior and the whole of the middle lobe sound. It is likewise only under certain conditions that circumscribed portions are attacked in the midst of the parenchyma, the intervals, together with the surface of the lung, remaining untouched. (Central, lobular pneumonia: see Bronchitis and Pneumonia Notha.) In these instances it is hardly possible to form a correct diagnosis, from physical signs alone. When both lungs are affected with pneumonia, the inferior lobe of the right lung is mostly found hepatized, and partially infiltrated with pus, and the upper lobes, and likewise the inferior lobe of the left lung, in the first stage of the disease.

One stage is invariably developed out of another. Thus, hepatization always begins at the centre of a patch in the first stage of inflammation, and spreads on every side towards the margin; meanwhile a sound neighbouring patch becomes involved in the first degree, and so on. Purulent infiltration, in like manner, always commences at the centre of an hepatized portion. The duration of the respective stages is indefinite; the third stage may, however, be attained within five or six days.

According to Louis (Recherches sur la Phthisie) pneumonia is more frequent in men than in women, (according to Pelletan, in the proportion of 10 to 1.) It would appear to be not less common to manhood and in advanced age than in youth. Out of 28 persons who died of pneumonia at the Leipsic hospital, 7 were under thirty, and 21 between thirty and sixty years of age. This would, strictly speaking, perhaps merely show pneumonia to be more fatal during the period last referred to; general experience, however, goes to prove that if we except old age, the disease is scarcely less fatal in early than in more advanced years.[1]

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[1] Cruveilhier states (livr. xxix,) that at the Salpêtrière, at Paris, five sixths of the old women die of pneumonia. Powerful predisposing causes to its development prevail at that institution.

Various general and local affections are peculiarly liable to associate themselves with inflammation of the parenchyma of the lungs, occasioning various deviations in its course, and especially in the character of its inflammatory product.

*Drunkards* are proportionately most prone to the disease,[1] and, what is remarkable, it scarcely manifests itself in them by the usual vital symptoms,[2] although spreading, and advancing to the last stage with astonishing rapidity.

Pneumonia very frequently supervenes upon *catarrhal* and *tubercular* disease of the lungs; in the former instance, as central (lobular) pneumonia, in the latter, overspreading a large portion of the lung without involving its totality. Hepatization only occurs in parts immediately adjacent to single tubercles, leaving crepitant air-cells everywhere in the intervals. (See Acute Tuberculosis.)

Pneumonia is observed as an accessory to *measles* and *smallpox,*—more frequently to the former, and in some epidemics it proves a dangerous complication. In measles, pneumonia is mostly central; in smallpox it occurs under various forms,—during the suppurative stage, sometimes as phlebitic lobular abscess. Inflammation of the substance of the lungs is mostly of a very insidious character, when supervening upon other diseases. Its first approaches are apt to remain hidden from the inexperienced observer, and its characteristic symptoms not to become manifest until it has reached a high degree of intensity.

*Typhoid disease,* although properly attended by mere stagnation of blood within the texture of the lungs, sometimes gives rise to real pneumonia. The substances effused are then but little plastic, of an unsightly dingy gray, as if mixed with decomposed blood. The hepatization is here extremely soft.

The inferior lobes are most frequently affected, and the right lung oftener than the left, as admitted by most pathologists. Out of 88 cases Andral found the inferior lobe diseased in 47, the upper lobes in 30, and the whole lung in 11. Out of 210, 121 were on the right, 58 on the left, and 25 on both sides. According to Chomel, out of 59 cases, 28 were on the right, 15 on the left, and 16 on both sides; in these latter, the right lung was more inflamed than the left. Of the 28 cases above cited (see p. 215), 16 were on the right, 7 on the left, (of these, 2 in the upper lobe and 1 central,) and 5 on both sides, of which latter, three were in a more advanced stage on the right side. These numerical relations are corroborated by Berg (l. c.), who met with 201 cases on the right, and 134 on the left side; and again by Pelletan, (Rev. Méd., Fevr. 1840, p. 284.)[1]

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[1] Berg, of Stockholm, has furnished us with some minute data concerning this and other complications. (See Schmidt's Jahrbücher, vol. xxvi, fasc. 3.)

[2] In this respect an excellent criterion may be derived from the researches of Lessing, who found that in delirium tremens, *without* chest affection, the proportion of the inspirations to the strokes of pulsation in a minute, was as 1 to 6·084, whilst in healthy persons it is as 1 to 4; and, on the other hand, in certain febrile diseases as 1 to 2, and upwards. (See Lessing, Diss. de inspirationum et pulsuum frequentiæ comparatione præcipue in febribus. Lips. 1838.)

The disease is fatal in every stage. There are examples of both lungs being found inflamed throughout, all three stages coexisting. In many instances, however, death ensues when but the half of one lung is in the first stage of inflammation. This is naturally dependent upon age, individual constitution, complication, and the amount of vital reaction present. According to Chomel, inflammation of the left lung, of the upper lobes, and in the female sex, is most apt to lead to a fatal issue.

The lobular pneumonia of children was first anatomically investigated and described by Guersent,[2] Leger,[3] and Billard.[4] Having examined many cases of the kind myself, I am nowise convinced that all the changes in the texture of the lungs comprehended under this category in new-born infants, are really the result of inflammation. Many of them would rather appear to belong to what E. Jörg[5] has more accurately described under the term *atelectasis* of the lung, a disease essentially distinct from the pneumonia of infants, or children. The difficult task of defining these two morbid conditions, and of assigning distinctive anatomical characters to each, will be attempted hereafter. To avoid repetition, therefore, the reader is referred to the chapter on *Atelectasis* of the Lungs.

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[1] The statistics of pneumonia have been exhausted by Grisolle. (Traité pratique de la Pneumonie, &c. Paris, 1842.)

[2] Dictionnaire de Médecine, vol. viii, p. 96.

[3] Essai sur la Pneumonie des Enfans. Thèse No. xlvi. Paris, 1823.

[4] Maladies des Enfans nouv. nés. Paris.

[5] Die Fœtuslunge im geborenen Kinde (the fœtal lung in the infant after birth). 1835.

The degeneration of the texture of the lungs which frequently follows phlebitis, and the absorption of pus consequent upon great surgical operations, &c., virtually constitutes true *lobular* pneumonia. Cruveilhier, Dance, and others give a very exact account of this kind of circumscribed pneumonia, whereof numerous instances have since been published: previously, attention had only been directed to lobular inflammation and abscess of the liver. Arnott and Albers have likewise furnished interesting details, concerning the origin and progressive development of this malady. The important anatomico-pathological relations which bear upon this subject, have been discussed under the head, *phlebitis*. We shall, therefore, here restrict ourselves to a description of the morbid state in which we find the lung itself.

In the cases in question we find isolated in the centre of sound lung, patches almost of a spherical shape, of the size of a walnut, and upwards. These are perfectly dense, non-crepitant, and of a straw-colour, or else of a dirty-brownish yellow. They have altogether the appearance of abscesses, but without containing fluid matter, their consistence being in general somewhat greater than that of portions of lung involved in the third stage of inflammation; although at the centre, or perhaps at a few other points, they are softer; indeed, according to some pathologists, they are capable of eventually liquefying altogether. Their cut surface appears homogeneous throughout, studded with little granular elevations; generally speaking, no fluid escapes when they are cut asunder. Within a circuit of a few lines from these patches, the pulmonary texture is in a state of red or gray hepatization; beyond this, it is perfectly healthy, only somewhat moister than usual. I have mostly found the branches of the pulmonary artery leading to the diseased parts, filled with consolidated blood, or with an adherent, faintly brownish coagulum. These purulent deposits (termed by Morgagni, to whom they were familiar, *tubercula*) occur either in both, or in one only of the lungs,—and several are more frequently met with than a solitary one; they are sometimes deeply seated in a pulmonary lobe, more frequently, however, near the surface, immediately beneath the pleura. They then press forward in some degree beyond the surrounding texture, often exciting a partial inflammation of the pleura, the latter either becoming invested with an adventitious membrane, or entering, at the implicated part, into a loose connexion with the costal pleura. The parts affected do not exhibit a purulent character at first, but are sometimes found in a state in which their several grades of development can be readily traced. Like the apoplectic clot, these patches consist, at the outset, entirely of coagulated blood, which, however, is rapidly decoloured, exhibiting internally gray points, which gradually spread, until the whole mass presently acquires the aspect of pus; the neighbouring pulmonary texture meanwhile passing into the second grade of inflammation. These lobular abscesses, however, only assume a yellow colour where the pus, taken up by the blood, is of a healthy kind. Where the pus is ill-conditioned and mingled with ichor, as in carcinoma,[1] the obstructed portions of the lung are of a dingy gray, or a pale-faint brown; the inflammation in the surrounding texture more intense, and the whole part much softer. These abscesses mostly destroy life within a short time; in rare instances, however, recovery is known to take place. Of this, a case of Oesterlen appears to be an example, (Heidelb. clin. Annal. vol. iii, fasc. 3): venesection had led to phlebitis, in the progress of which, amongst other menacing symptoms, an abscess formed in one of the axillæ, and purulent expectoration from the lungs followed. Complete recovery did not take place until after the lapse of five years; the disease having meanwhile long simulated pulmonary phthisis. Occasionally, perhaps, these lobular abscesses become encysted within the lungs, undergo scar-like shrivelling, and are transformed into hard calcareous concrements.

*Hypostatic pneumonia*, first pointed out by Piorry, (Clin.

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[1] The interesting researches of B. Langenbeck (Schmidt's Jahrb. d. ges. Medic. vol. xxv,) show that lobular abscesses resulting from cancerous destruction, differ essentially from those produced by simple absorption of pus. In the instance of the former, real cancer-cells, or their nuclei, enter the veins, and passing from thence through the right side of the heart and the pulmonary artery, into the capillaries of the lungs, where they lodge, owing to their size, just like the pus-globules in phlebitis (which see). These cells mostly excite in the pulmonary texture a fatal lobular inflammation (in which they themselves merge), and cause death. In the lungs the obstructed cancer-cells rarely (in Langenbeck's experience only once) form the seminal principle of secondary cancerous growths.

Méd. de la Pitié et de l'Hospice de la Salpétrière, &c. 1833,) chiefly befalls aged persons, and is characterized by the peculiarity of its development and diffusion, as also by the irregular and insidious nature of its course. Here the disease does not always stand connected with the boundaries of lobules and lobes, so that we could say—this or that lobe is pre-eminently its seat; it affects the posterior surface of all the lobes, penetrating from thence to the depths of the lung. Ordinary pneumonia advances from lobule to lobe, &c. in masses; hypostatic pneumonia is wont to spread in the direction of the surface. The most deeply situate portions of the lungs are here the most intensely inflamed, being found generally, in the second, less frequently, and only partially in the third grade. In other cases the texture of the lungs is mostly affected, about the roots of the bronchia and blood-vessels.

This *senile* pneumonia has been far more carefully investigated by Hourmann and Dechambre, (Arch. génér., March, September, and October, 1836,) than by Piorry,[1] so that, in pointing out the distinctive features of this form, I shall chiefly adhere to their description. The differences are mainly dependent upon the peculiarity of structure of the lungs in old age, for an acquaintance with which we are indebted to the aforesaid pathologists.

The *first stage* is nowise remarkable, except that, in dried lungs, it is easier to study the minute capillary injection of the parietes of the pulmonary cells, which, in the aged, are always considerably more capacious than in the young.

In the *second stage*, essential deviations occur. The inflamed lungs are never so weighty, nor so excessively distended and enlarged, as in ordinary pneumonia; the involved portions do not entirely sink in water, but remain suspended, although at a considerable depth. *Two forms* of this stage are to be recognized in the aged. The first is characterized by the lung, on incision, appearing perfectly smooth. The cut surface is homogeneous, of a very dark colour, and allows a viscid, but frothless reddish fluid to ooze away. The patches, thus impermeable, are sometimes elastic; sometimes quite softened. In the second form the cut surface is certainly granular; but the granules are invariably much larger than in the hepatized lung of younger individuals; they are sometimes regularly spherical, and uniform in size, at other times extremely unequal, both in shape and circumference, according as the senile condition of the parenchymatous structure is more or less advanced. The hepatized patches are seldom in an equal degree softened, although always moister than in ordinary pneumonia.

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[1] Arch. gén., Août 1835. Compare the chapter on Emphysema of the Lungs.

In the *third stage* we have a recurrence of the above two forms. In the first place the cut surface is even, presenting irregular, narrow streaky spots of puriform matter, which, by moderate pressure, may be made to exude. When portions, thus drained, are dried, the lungs are found again thoroughly permeable. In the second form the cut surface offers the same more or less coarse granulations as in the second stage; the softening is here, however, at least equivalent to that of ordinary pneumonia.

Finally, there still remains a *variety* of the third stage, first described by Hourmann and Dechambre, confirmed by Prus (at the Bicêtre), and twice observed by myself. The purulent matter is pretty sharply defined in spots, from one to two lines in diameter. Its consistency is very slight, its colour, resembling faded ivory; it is easily removed with the point of the knife, when it is found to have occupied those irregularly dilated air-cells, often met with at intervals throughout the lungs of aged persons.

The two forms described in the two latter stages, constitute, according to Hourmann and Dechambre, the distinctions between interstitial and vesicular pneumonia; but, although their reasoning in support of this view appears well founded, the question is still in need of further confirmation. I must, however, remark that the smooth hepatization above mentioned has occurred to me not merely in the aged, but likewise in persons who have been long bed-ridden, from some disorder involving great prostration of nervous energy, (as apoplexy, palsy, and the like,) and at length die, under symptoms *apparently* catarrhal, but perhaps *really* due to inflammatory irritation, resulting from genuine hypostasis of blood in the lungs. In all these instances only one lung was so affected, the other exhibiting simple stagnation.

In senile pneumonia the *bronchial* mucous membrane is always much reddened, and the air-passages, from the trachea to the minutest bronchial ramifications, replete with a turbid, tenacious mucus. Piorry appears to have erred in stating the *pleura* to be rarely implicated; Hourmann and Dechambre having found pleuritic false membranes of recent formation, in 38 cases out of 60.

We must guard against confounding hypostatic peripneumony, with that condition frequently encountered in the lungs of persons who die of typhus. There the lung is likewise of increased gravity, little permeable, and mostly softened. On careful comparison, however, it will be seen that these changes are the result of stagnation of, for the most part, diseased blood. The lung is not distended beyond its usual volume, but rather collapsed; its substance being still filled with fluid blood, and, through imbibition, stained of a blue-black. By careful ablution, the stain cannot indeed be removed, but the substance may, in some measure, be restored to its natural state, whilst in the texture so altered, no vestige is ever seen of any inflammatory product. This stagnation of blood is always found equably diffused along the posterior surface of the lung, whilst the anterior half of the lung is generally observed to be bloodless, dry,—but perfectly sound otherwise. It is seen almost in an equal degree in both lungs, and appears to be the effect of dynamical as well as mechanical influences, the former consisting in disorder and gradual withdrawal of innervation through the pneumogastric pair of nerves. This subsidence of the blood is, therefore, met with in all diseases, wherein the primary or secondary cause of death is, as it is commonly said, in the brain, and its extent is proportionate to the duration of the agony. It is, for mechanical reasons, easily intelligible, that it *must* occupy the posterior part of the lungs. Finally, the consolidation of the pulmonary texture consequent upon effusion within the thorax, is of a purely mechanical nature, and has already been described under the head of pleurisy.

The anatomical relations connected with the *resolution* of pneumonia, are extremely difficult to ascertain, opportunities of examining the bodies of persons who happen to die of another disease during convalescence from pneumonia, being very rare. Scarcely an instance of the kind is upon record. Laennec states, that when resolution occurs during the first stage, the infiltration of blood is certainly removed by absorption, but the lung long continues heavy and imperfectly elastic, the parenchyma being loaded with serum. In the second stage, the hepatized portions are paler, and remain long of a reddish gray before they resume their natural colour; at the same time they become moister, from liquefaction of the coagulated effusion within the pulmonary cells, (the expectoration at this epoch acquires almost a purulent aspect;) the hepatized part gradually, and proportionately to this liquefaction, becomes again pervious to the air, but for a long time continues heavier, denser, and moister. In the third stage, the yellow colour of the lung, having previously assumed a greenish cast, becomes fainter, the pus mingles with serum, diminishes, and, in a corresponding measure, the air re-enters. Last of all, a dusky, dirty yellow colour remains, the texture becomes still moister, but pus no longer exudes. In every instance an undue proportion of mucus long continues to be thrown out from the air-passages, and the secretion is only reduced to its proper amount and character, by slow degrees. In certain cases the air-cells, injured by inflammation, do not recover their normal function—(the secretion of elementary molecules,)—the effused substances do not liquefy, but become amalgamated with the parietes of the air-cells; and thus the part remains impervious. This obliteration of the hepatized structure is connected with vascular development in the effused product; it is, however, uncertain whether this act of organization be due to the pulmonary or to the bronchial arteries. The consequence seems to be permanent incapacity of the part for the office of respiration, gradual shrivelling of the pulmonary texture, sinking in of the wall of the thorax, and dilatation of the bronchial tubes throughout the previously hepatized lobe. (See Bronchiectasis.)

## SECT. II.—CHRONIC PNEUMONIA.

The occurrence of chronic pneumonia, as also the possibility of demonstrating it anatomically, have been doubted by many pathologists, notwithstanding the repeated statements of Andral as to the reality and frequency of this disease. The truth is, that it is not common,—its development depending upon a rare concurrence of circumstances.

It scarcely ever arises out of ordinary acute pneumonia, but coincides, most frequently, with the formation of tubercle. It is probably never idiopathic, but rather an accessory to certain other diseases. It is difficult to determine what is the ground of distinction between chronic and acute pneumonia;—indeed, the nature of chronic inflammation appears to be altogether ill understood, for slowness of progress and diminished intensity are not its sole characteristics. In the few instances in which chronic inflammation occurs as a direct sequel to acute, there is an inordinate accumulation of morbid products, which can only be assimilated or otherwise removed, through the sustained influence of augmented vascular action; in others it is the abiding influence of an unusual morbific cause, grafted upon the previous one; in others again, it is a peculiar diseased tendency, to which the development of the morbid product is due, and which occasions the commingling of the genuine deposits of inflammation with alien substances, whose presence keeps up a severe, if not insuperable, degree of irritation. Sometimes the inflammatory state is, so to speak, from the beginning chronic, more especially where, owing to something anomalous in the process of nutrition, heterologous growths form at the expense of the natural organic structure. If, together with this, we take into account the modifications resulting from locality, (see Pleurisy,) or from the kind and degree of vital reaction in the implicated organ, it is obvious that chronic inflammation is by no means so simple or readily understood as the ordinary acute kind, that almost every individual case forms in itself a variety, and that it is of the utmost difficulty to determine the exact line of transition from the acute into the chronic state. From these remarks, all more or less applicable to chronic pneumonia, it may be inferred, that an ordinary inflammation may sometimes become still more protracted in its course, than certain cases reputed chronic.

According to Andral, most varieties of this disease are distinguished by the texture of the lung not being softened as in acute pneumonia, but on the contrary hardened. Two forms are discriminated, namely, a red and a gray induration. It is difficult to say whether, in all cases, these two are gradations only, as Andral's description seems to indicate, or whether the colour is the result of other accidental circumstances, as Hope maintained. If we recur to the observations before made, and at the same time reflect that those two colours are by no means the only ones, but merely the types of numerous shades and varieties, Hope's opinion must appear the more generally correct of the two. Besides the above principal forms, Andral assumes a third, namely, black induration,—thus classifying under chronic pneumonia that transformation of the substance of the lungs, which some pathologists, and Laennec in particular, designate as melanosis, others again as the result of black pigment, deposited in excess, owing to some peculiar morbific action. But this would be giving too wide a range to chronic pneumonia.

We meet most frequently with the gray induration, or, as it is sometimes called, white hepatization. It is mostly associated with the development of tubercle, and in the few marked instances which came in my way, I was unable to draw the precise limits between pneumonic induration, and gray tubercular infiltration. The former was distinctly cognizable in the lower half of the lung, the latter in the upper half; at the apex, were several small cavities, precisely resembling those of tubercular phthisis. There were at the same time traces of the tubercular constitution in other organs. The transitions and combinations of the two diseases are probably numerous, whilst chronic pneumonia is sufficiently rare to render the discrimination in a given case a difficult task. At the part affected, the lung is distended, preternaturally heavy, and completely impermeable to air; even the bronchial twigs are mostly choked up with a solid whitish matter. (Compare Gendrin, l. c.) Of the natural texture, nothing is distinctly traceable; the surface on incision looks gray, here and there inclining to yellow, diversified with separate white stripes and arborescent patches of black pigment. The whole mass is hard, incompressible, neither friable nor easily penetrable by the finger, but yet fragile and readily torn. The cut surface is smooth, and even devoid of the granulations of acute pneumonia. Nothing exudes on incision, and but a small quantity of turbid grayish fluid on pressure. It is mostly the inferior lobe of one or both lungs that is thus affected, and as the chronic inflammation proceeds from below upwards, it meets the tubercular process advancing in the opposite direction. In the examples seen by me, the chronic pneumonia involved the lower portion of one lung only, whilst the tubercular affection was seated in both.

Different circumstances may tend to modify the anatomical character of chronic pneumonia. The colour—varying according to the nature of existing complications, the more or less rapid course of the disease, and the age of the individual—is either of a dingy reddish, brownish, pale violet, yellowish white, iron gray,—or nearly white. In children who had shown symptoms of inflammation, I have sometimes found a light gray, almost white, or yellowish induration of a whole lobe or of certain lobules, and more frequently of the upper than the inferior. This lobular induration must not be confounded with the swollen, tuberculous bronchial glands, which, in young children, are often found deeply impacted within the pulmonary substance.

It would be very difficult to demonstrate whence it happens that, the external circumstances being very nearly identical, we have, in one case, the chronic pneumonia just described, conjointly with tubercular development; in another, acute tuberculosis; and again, in a third, ordinary phthisis pulmonalis. I submit the following explanation, without, however, intending to attach to it more weight than belongs to hypothetical reasoning in general. In the variety of chronic pneumonia just alluded to, the inflammation is purely local, and first calls into activity the tubercular predisposition, which has not yet taken sufficient hold upon the constitution to display itself in any particular part, without some adequate impulse. Now, a morbid product of inflammation effused under the above circumstances, carries within it the germ of tubercular development; thus its effect is different from that of effusion purely inflammatory; for it acts as an abiding source of irritation, keeping up the disease and promoting its further development. In acute phthisis, on the other hand, the tubercular diathesis is present in an exalted degree, the whole œconomy, particularly its fluid parts, being, so to speak, saturated with the morbific elements. Under these circumstances, a febrile movement, however induced, immediately determines a general morbid action, and tubercular matter is copiously thrown out upon the organs most prone for its reception,—namely, the lungs. Every sudden deposition of extraneous matter necessarily excites inflammatory reaction in the organs concerned. Ordinary pulmonary consumption, again, develops itself slowly, under the progressively increasing influence of the tubercular cachexia, or proceeds from one of the more acute forms before mentioned. Careful comparison of the anatomical and pathological characters of these three varieties of disease, favours the above explanation.

In chronic pneumonia, other heterologous substances, medullary fungus in particular, enter into the same reciprocity of action with the inflammatory product, as tubercle. (See Cancer of the Lung.)

Andral's second form, *brown induration,* is far more rare, and apparently not necessarily connected with tubercular or other cachexia. Its course is more acute, and the parenchyma of the lungs is moister and less compact than in the gray induration. I have met with it several times as a consequence of extensive hypertrophy of the heart; the inferior lobe (of the left lung more especially, though occasionally of the right) being each time implicated. The chronic inflammation appeared to have sprung from simple congestion; the loaded state of the pulmonary vessels, common in heart disease, being here, not merely kept up, but even exalted to an inflammatory pitch by the uninterrupted influence of morbidly increased impulse of the heart. The diseased lobes were but moderately distended, which is easily explained by the tendency of the hypertrophied heart to displace the neighbouring organs; their colour was a dark brownish red, both their absolute and specific gravity augmented, and their consistency greater than usual. Their parenchyma appeared consolidated, nowhere permeable, nowhere crepitant, nor yet saturated with blood, as is common in heart disease,—and contained only a moderate quantity of serum. The cut surface was smooth and even. In one case there were, in the midst of the brown induration, little isolated patches of a faint yellow hue, a good deal softened, and sharply defined. These portions evidently denoted a higher grade of inflammation, inclining to purulent formation.

Andral and Hope describe chronic pneumonia as a slowly-developed hypertrophy of the septa of the lobules and cells, attended, during the period of augmented vascular activity, with a very gradual deposition of albuminous matter into the interstices of the pulmonary substance. In some instances the cellular septa of the pulmonary vesicles are said to increase in consistency, so far as to acquire a semi-cartilaginous character.

According to this view, gray and red induration should consist in an inflammation of the interstitial texture, which is possibly the truth; for the parietes of the pulmonary cells, irritated by the presence of a peculiar inflammatory product, displays considerable vascular activity; and thus the whole pulmonary texture unites with the effused substances, degenerates, and eventually forms with them a single, tolerably congruous mass. The bronchial twigs are compressed, whilst the pulmonary vessels frequently contain fibrinous plugs more or less solid, and coagulated blood. Hence, exudation into the cavities of the vesicles, precedes the swelling and hardening of their parietes.

Other organs suffer only in so far as some complication or constitutional disorder is either the source or the consequence of the inflammation.

## SECT. III.—PULMONARY ABSCESS.

At the present day those collections of pus which result from pure inflammation of the parenchyma, are alone designated by the above name; at an earlier period, pulmonary abscess was reckoned a very frequent phenomenon. The truth is, however, that imposthume was then confounded with gangrenous destruction, with the accumulation of muco-purulent matter in dilated bronchia, with the lobular abscesses consequent upon phlebitis, and with cavities filled with softened tubercle.

Abscesses within the texture of the lungs are very rare as a sequence of ordinary pneumonia. Laennec met with but five or six examples; Andral (Clin. Méd. ed. 4, vol. iii, p. 468) with four; Gendrin with two; Honoré, Bouillaud, Becker, Arnsohn, and Heyfelder,[1] each with one. Hourmann and Dechambre (Arch. gén. 1836) found abscesses in two out of 88 cases of pneumonia; one of them, of enormous size, occupying more than a third of a lung. In children pneumonic abscess has been witnessed by Hope (Principles of Path. Anat.), Andral (l. c. p. 468, foot-note), Billard (Traité des Maladies des Enfans nouveau-nés, p. 516-18), and others. The character of the cellular texture of the lung is obviously unfavorable to the formation of abscess. There is, upon the whole, very little loose cellular texture in that organ, which presents the greatest possible development of surface within the smallest compass; that little is very tense, and only in delicate layers, so that it is seldom possible to inflate even small portions of it. (Compare Cerutti's 'Collectanea quædam de Phthisi,' &c. Lips. 1839, p. 16). As the multitude of air-cells offer ample room for the lodgement of pus, and as a long period must elapse, ere the solid and elastic interstices are destroyed by softening, true abscess seldom forms, while purulent infiltration, the third stage of pneumonia, is comparatively frequent. Hence Laennec very justly observes, that patients generally recover from pneumonia of limited extent, but when it is more diffused, die, before the pus can collect into an abscess. I have often found portions of lung in the third stage so thoroughly softened, that the mere accidental pressure exerted in removing the thoracic viscera, or an intentional thrust with the finger, gave the semblance of a veritable abscess.

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[1] Studien im Gebiete der Heilkunde, vol. i, p. 52; containing an elaborate analysis of a number of cases, with especial reference to diagnosis.

Purulent accumulations within the lungs vary in circumference, from that of an almond to that of a man's fist; the majority are, however, trifling. They have no sharp outline, but are lost, as it were, in the surrounding texture. They are, therefore, not inclosed either within walls presenting a definite surface, nor within false membranes; on the other hand, we find the pus the more consistent, the farther it is distant from its centre; it then merges in a sloughy pulp, and this again in a pus-infiltrated texture, which gradually blends with the natural substance of the lung. Gendrin once met with an elongated deposit of pus of the size of the little finger, with a jagged border, and in communication with a bronchial tube of the thickness of a crowquill. This was cleft throughout its length, so that its inner surface formed one of the walls of the abscess. As a general rule, pus forms most readily in the vicinity of the bronchial tubes, where the cellular texture is most abundant and loose. Gendrin and myself frequently found fluid pus around and along the walls of the bronchial tubes, in the third stage of pneumonia. It is conceivable, as stated by Carswell (Fasc. Pus, pl. ii, fig. 4), that it may in like manner often accumulate within the cellular sheath of the pulmonary vessels. Pulmonary abscesses are seated for the most part in the inferior lobes; in one solitary instance (Heyfelder) in the superior, and in two others (Laennec, Heyfelder) in the middle lobe. There is in general but one present. An instance of several occurring in one (the right) lung, is related in the 'American Journal of Medical Science:' an iron nail fell into the trachea of a girl of 5 years, whilst at play, and the child died after the lapse of a twelvemonth; on cadaveric inspection, the nail was found impacted in the fourth or fifth division of the right bronchus, the lung containing several deposits of pus, communicating with the bronchial tubes.

Earlier writers record instances of these abscesses penetrating through the intercostal muscles, or through the diaphragm, into the liver, &c., or of their reaching the pleura, and establishing empyema. In all probability, however, other morbid conditions have been mistaken by them for pneumonic abscess. Care must be taken not to confound veritable abscess of the lung with those accumulations of pus, stated by Hope and Andral to be frequently met with in the pulmonary arteries and veins. In the third grade of pneumonia, purulent fluid has been discovered even in the lymphatic vessels of the lungs.

*Cicatrization of the abscesses* has been too seldom witnessed to be described in detail. Laennec mentions in general terms that pulmonary abscesses heal in the course of from six weeks to as many months. Sometimes, however, Nature's efforts are inadequate, and the cavity remains open for years. Thus in a case related by Becker (Preus. Vereins-zeitung, 1834) the cavity of an abscess remained unclosed for nine years after the date of the pneumonia. This cavity discharged its contents daily, and its internal surface was found coated with a mucous-like membrane, surrounded by indurated pulmonary substance. In general, where the healing process commences, the purulent destruction is arrested, by the neighbouring parenchyma hardening to a cellulo-fibrous crust, and collapsing; the bronchial branches involved are at the same time obliterated, and at their blunt extremity dilate into a pouch. On one occasion I found the whole of the middle lobe, and on another a patch, the size of a man's fist, of the inferior lobe, transformed into a thoroughly dense mass of almost cartilaginous hardness, and surrounding, to the depth of upwards of an inch, a cavity replete with muco-purulent fluid. Several bronchial branches, greatly dilated, opened into the cavity, the internal surface of which was rough. In these 2 cases there had been, 5 and 9 months previously, severe and fatal pneumonia, which lingered on under continued fever, with several violent attacks of hæmoptysis intervening.

# CHAPTER III.

## DISEASES OF THE LUNG NOT NECESSARILY DEPENDENT UPON, OR ALLIED TO, INFLAMMATION.

### SECT. I.—GANGRENE OF THE LUNG.

Gangrene of the lungs, though more frequent than abscess, is a rare affection. It occurs not only as an unusual termination of pneumonia, but likewise independently of genuine inflammation of the lung. Towards its production must concur a peculiar disposition of the solids and fluids, dependent upon a particular nervous influence, or else upon a total cessation of nervous action in the diseased part. Schönlein has designated the characteristics of this morbid process, generally, under the term *Neuro-phlogosis*. A part of the lung is gorged with blood, and the adjacent branches of the pulmonary artery are plugged with coagula. Hence results, in the first place, such a degree of pressure upon the entire mass of the complex texture of the lung, as to suspend all nervous energy: in the second place, a total absence of circulation to and from the part, cutting it off from its organic union, and necessarily leading to its death. Here the blood is in a condition very different from that attendant upon inflammation. Effusion is followed neither by suppuration, nor by simple softening; but by dissolution of what is fluid, softening of what is solid, and general decomposition,—vital reaction being, not merely modified, but directly suppressed. The great influence of the peculiar relations alluded to, may be inferred from the fact that, in gray hepatization, where the circulation is in like manner suspended, resolution usually takes place without any sign of gangrene.

This event may be a sequel to excessive inflammation; the pneumonia, however, in such instances, is limited in extent, and never involves the whole, or even the greater part of the lung. At the centre of the hepatized texture, instead of suppuration being established, a portion becomes perfectly liquescent, exhibiting a slate-colour, and emitting a fetid smell.[1] Gangrene may occur at various portions of an inflamed lung, towards the middle,—or near the surface of a lobe; it does not readily become isolated; at all events, it is difficult to discover any distinct boundary; for in the midst of a greenish, fetid mass, filamentous shreds of a substance partaking more or less the process of destruction, are seen hanging, as it were, by a pedicle from the pulmonary vessels. The vascular ramifications in the surrounding pulmonary texture, are found obstructed with a very dirty, yellowish plug, not always made up of concentric layers, as in the ordinary inflammation of blood-vessels, but rather a uniform pap, not reaching much beyond the gangrenous part. Sometimes, indeed, it extends to the principal branches of the pulmonary artery, though probably not till the approach of death. The bronchial twigs, on the other hand, project with open orifices into the sloughy mass, which, through them, may, under propitious circumstances, be entirely discharged,—and a cure effected. This favorable termination is rare, for pneumonia, issuing in gangrene, proves rapidly fatal, owing to the great extent of the organic mischief. Occasionally, however, life is prolonged for a fortnight or six weeks beyond the first commencement of the gangrene.

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[1] When the pneumonia has already attained its third stage, the gangrene is of a dirty gray tint.

Gangrene resulting from *chronic pneumonia* is uncommon; it has been observed only by Andral (l. c. 4 éd. vol. iii, obs. 64) and Carswell. The gangrenous cavities occurred in the midst of a pulmonary lobe, in the state of red induration, and were already isolated by the intervention of a thin layer of a greenish substance. It is still a question whether, in such instances, the gangrene may not have originated independently, and the surrounding inflammation have been secondary. Carswell supposes that where chronic inflammation precedes,—the death of the gangrenous part is brought about by obstructed circulation, the blood-vessels being compressed and obliterated by the indurated texture. His figure (Fasc. Mortification, pl. iii, fig. 4) would certainly show this to be so, provided we have no grounds for believing the obliteration of those vessels to be, at least in some instances, the *consequence* of the gangrene. Perforation of the lung and of the pulmonary pleura by empyema, and also by vomicæ in phthisis, is likewise explained by Carswell as dependent upon chronic inflammation and consequent circumscribed gangrene.

As an independent disease, gangrene of the lungs has been met with more frequently, as may be inferred from Gerhard's numerous observations,[1] referrible for the most part, to persons whose constitution was destroyed by the prolonged abuse of spirituous drinks, by misery and by want, when otherwise exempt from organic disease. Moreover, the scattered examples recorded elsewhere, always relate to cachectic individuals, enfeebled by bygone distempers. Cruveilhier directs attention to the frequency of gangrene of the lung in epileptic subjects. The insane[2] appear, in like manner, to be particularly prone to the disease, more especially where the bodily health has suffered from neglect of cleanliness and the want of proper attention to the person in other respects, or where (in maniacs for instance) a greatly depressed state of the animal functions has succeeded inordinate nervous excitement. Sometimes the disease is caused by a septic agent directly tainting the body. Thus, in Carswell's case (Fasc. Mortification, pl. iii, fig. 5,) the sting of an insect brought on gangrene, first of the lip, and eventually of the lungs. In similar instances, the attack was consecutive to phlebitic lobular abscess. Pulmonary gangrene has been observed at every period of life, except the infantile. It would seem, however, to evince a preference for persons beyond the middle age, and for the male sex.

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[1] Annales de Médecine Belge et Etrangère, Août, Sept. 1838. The cases were collected at the hospital at Philadelphia.

[2] Guislain (Gaz. Méd. 1838, No. xxviii,) witnessed, within a brief period, fifteen cases at the hospital for insanity at Ghent. Three examples occurred at the Bicêtre within four months.

Laennec distinguished two forms of pulmonary gangrene, the *diffuse* and the *circumscribed,* and we are indebted to him for our first acquaintance, both with the signs and symptoms during life, and with the anatomical characters of the disease. The best account of *diffuse,* pulmonary grangrene, is that of Schröder van der Kolk. (Observationes anatomico-pathologici et practici argumenti, tom. 1, p. 202.)

In one instance, this pathologist found the lung adherent to the costal pleura, and, in the act of separating it, a portion of the inferior lobe was torn off, and gave escape to nearly a pound of blackish fluid, closely resembling that vomited in cancer of the stomach, only more offensive. In this and another case, he carefully injected the pulmonary arteries and veins, and found most of them obstructed to near the spot where the last flocculi of pulmonary parenchyma protruded into the gangrenous ichor, a few only admitting the injecting fluid to their minuter terminations. The lymphatic vessels received a portion; and the bronchial glands, which communicated with those lymphatics, were found much swollen, of a dingy gray colour, and thoroughly softened; showing, it would seem, that the gangrenous fluid had been actually absorbed. The walls of the gangrenous cavity appeared as if long macerated. The remainder of the pulmonary substance was sound, without a trace of inflammation or tubercle,—but moister than usual, less crepitant, and pervaded by a thin, turbid fluid. The pleura in the neighbourhood of the gangrenous part was thickened, of a dirty gray hue, and in some degree softened, or friable. According to Cruveilhier, (livr. xi, pl. 4,) there is, in *diffuse* gangrene, always some discoloured purulent fluid in its cavity; moreover, the sloughing process is apt to perforate the pleural investment of the lung, unless prevented by antecedent adhesions.

*Circumscribed gangrene of the lung* has three distinct stages: the formation of a slough,—its liquefaction,—and the establishment of a gangrenous cavity. Laennec, Andral, and Cruveilhier, have furnished the best anatomical descriptions of this form, but its symptoms and pathogenetic relations are not fully made out. *The sloughs* form either near the surface, or in the centre of a lobe of the lung. In the former case, the disease generally proves fatal under pleuritic symptoms, unless guarded by the rapid formation of sufficiently firm adhesions. The sloughs are of irregular shape, blackish-green tinge and moderate consistency. In colour they resemble, in some degree, those produced upon the skin with solid nitrate of silver; they are, however, much moister, and emit a very fetid smell. Sometimes they are surrounded, for half an inch in depth, with hepatized, greenish-gray, pulmonary texture, which is essentially distinguished from pneumonic hepatization by its humid character. In general, however, the adjacent portion is only softened, and infiltrated with a turbid discoloured serum, which, farther off, becomes more and more limpid. Cruveilhier terms this gangrenous œdema, and compares it with the œdematous swelling of gangrenous limbs. The implicated lobe of the lung does not collapse on the thorax being opened, but remains expanded; its gravity is, at the same time, preternaturally augmented. As soon as the slough is cast off, it becomes surrounded with a gangrenous ichor, which is occasionally saparated from the sound texture, through the intervention of a dirty green-coloured, loose, membranaceous layer. Generally, however, the gangrenous portion is not wholly detached, but is gradually resolved into a black-green pulp, consisting of fibrous detritus and a dirty-green ichor of a most offensive odour, in which scattered shreds hang suspended by a sort of pedicle from branches of the pulmonary artery. The vessels within and around such a patch, for the most part become obliterated, but now and then remain pervious; branches of sound artery have even been seen to traverse the gangrenous cavity. When, however, they suffer erosion before becoming thoroughly plugged,—violent, and in most instances fatal hemorrhage ensues. The liquefied mass escapes as blackish-green, very fluid and fetid sputa, the bronchial twigs, as already stated, presenting abrupt, gaping orifices to the cavity. Where the gangrenous destruction spreads towards the surface of the lung, either the latter unites very firmly with the costal pleura, or else the gangrenous ichor breaks through the pleura, (Gerhard, 7th case; Bouvier, Rev. Méd. 1838,) determining empyema and pneumothorax, or possibly fatal hemorrhage in this direction, (Cruveilhier, livr. iii, Pl. 2.)[1] If the gangrenous matter has been removed through the bronchia, and nature has set limits to the destructive process, we find, in some instances, a tolerably smooth cavity lined with adventitious membranes. The walls of this cavity are dark-red, at a later period rather of a yellowish-white, and of considerable firmness; they adjoin a softer, discoloured, and very moist texture, which latter again is surrounded by healthy parenchyma. A cavity of this kind may exist for a long period after all gangrenous affection has ceased, without thoroughly cicatrizing. Gerhard, (case 5,) had an opportunity of examining the body of a man who had been under treatment for pulmonary gangrene nine years previously. He found the inferior lobe of the left lung most firmly adherent to the costal pleura, very lax, and of very small dimensions. In the vicinity of the larger bronchial branches of this lobe, was a shrunken, flattened cavity, an inch long and equally broad, which communicated with a bronchial branch. This cavity was invested with a lining membrane, in habitude closely resembling the mucous membrane of the smaller bronchia. The first sign of *cicatrization,* apparently a very rare event, proceeds from the part immediately surrounding the gangrene, which gradually recovers its consistency and permeability; meanwhile the infiltrated fluid is gradually absorbed or expectorated, and the gangrenous patch becomes sharply circumscribed, or cut off, so to speak. (Cruveilhier, livr. iii, pl. 2.) The fetor now ceases, and the secretion becomes more puriform and less copious. The purulent false membrane, lining the cavity,—at first soft, becomes consolidated, and, as I have myself ascertained in one instance, acquires blood-vessels of its own. This thorough depuration of the affected part from all diseased texture, occurs, however, very seldom. More frequently the whole circumference of the cavity is reduced to a compact, grayish-white mass, which closely adheres to the walls of the thorax, forming a junction with its blood-vessels (branches of the intercostal arteries and of the internal mammary artery), and assuming generally the attributes of a cicatrizing tubercular cavity.

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[1] Hughes (Lond. Med. Gaz. May 1837, p. 303,) describes a case in which a violent blow inflicted on the body gave rise to pulmonary gangrene, and the destructive process eventually extended to and perforated the diaphragm.

Where the gangrene is not distinctly circumscribed, the destruction spreads, and forms a veritable ulcerous patch, the discharge from which continues ichorous. The parietes of the cavity do not become clean, nor smooth, but remain uneven, with projecting flocculi, and with furrows, fissures and sinuses, which penetrate deeply the surrounding texture. In this manner, diffuse, is sometimes developed out of originally circumscribed gangrene. The mucous membrane of the bronchia, to the furthest ramifications of the air-passages, is deeply reddened and softened in consequence of the frequent contact of the decomposed matters.

Circumscribed gangrene occurs in all parts of either lung,—though more especially in the inferior lobes, and in the right lung. There is mostly but one gangrenous cavity; where there are several, the destruction is of purely septic origin.

Finally, in some cases of tubercular phthisis, the expectoration assumes a gangrenous character, certain portions of lung sphacelate, partially separate, and are coughed up, or else gradually liquefy in the tuberculous cavity, emitting a very offensive odour. This is probably a sequence of the sudden and extensive closure of the pulmonary arteries. (See Tuberculosis.) Stinking expectoration alone does not, however, always denote the existence of gangrenous destruction, either in phthisis or in chronic catarrh.

## SECT. II.—ŒDEMA OF THE LUNG.

The cellular and eminently vascular parenchyma of the lungs appears to be quite as prone to serous infiltration as the subcutaneous cellular texture, if not more so. The same causes apply equally in both instances, and may be purely mechanical, or of a dynamical character, and the latter again either of a passive, or of an active kind. To discriminate in these respects in a given case, is of the utmost importance, with reference both to treatment and to prognosis. The task is, however, sometimes extremely difficult, owing to the multiplicity of special etiological relations, and of transitions from one form to another, particularly from mere passive to active œdema, and from thence to congestion and inflammation of the lung.

In mere *passive œdema*, the pulmonary texture is heavier and somewhat increased in volume, although by no means so much so as in pneumonia. Crepitation is diminished, the lung feels like a sponge saturated with water; its elasticity is greatly impaired, and it pits upon pressure, almost as is the case in anasarca. The texture is, however, by no means softened, being still possessed of all its natural tenacity. The lung is pale, grayish, with a shade varying from yellow to blue, but presenting something peculiarly clear and limpid in its aspect. On incision, a vast quantity of a colourless, sometimes inclining to yellow, limpid fluid readily escapes, appearing to ooze equably from the whole surface, and exhibiting a slight degree of frothiness, which is more distinct in proportion as the œdema is of recent date. The fluid is but sparingly mixed with blood, from the vessels implicated in the incision; for, in mere passive œdema of the lung, both the smaller branches and the capillaries are exsanguineous. Serous infiltration appears to occupy not only the pulmonary cells, but likewise the interstitial cellular texture. It is, therefore, almost impossible to recognize the parenchyma of the lung at the first glance; the pulmonary cells seem to have vanished, and do not become distinct again, even after all the fluid has escaped; excised portions of lung are lax and flabby, do not crepitate, and sink completely, or in great part, in water.

Passive œdema of the lung, in a slight degree, is extremely common in subjects who have died of very different maladies, being in most instances probably developed during the agony. In the rare cases in which it occurs as an independent disease, it invades both lungs, and is not always governed by the laws of gravitation; for I have frequently found it in a far higher degree in the upper lobes, and near the anterior surface of the lungs, than at their posterior part. In a very few instances it operates as the solitary cause of death,—suddenly putting on a violent character, and being now and then accompanied by hemorrhage. It might then be denominated serous apoplexy of the lungs.

There is hardly a single disease of the lungs or heart which is not, prior to its fatal termination, more or less complicated with pulmonary œdema. The latter does not then, however, make its appearance exactly as above described, the organic changes peculiar to those several diseases being simply modified by the extraordinary serous infiltration, and one characteristic feature of idiopathic œdema,—namely, the anæmious condition of the pulmonary texture,—being for the most part entirely absent.

A most important feature in a practical point of view is this, that during the progress of, and more especially the convalescence from pneumonia, an excessive amount of serous fluid is thrown out into the diseased lung, if not into both lungs. The pneumonic symptoms are thus essentially modified, and the whole course of the affection, or at least the period of convalescence, protracted,—nay, a fatal issue may be the result. The same thing often happens in bronchitis. It may be here remarked, that serous infiltration of the pulmonary texture may be reckoned in many instances as the immediate effect of a limited degree of inflammation, just as the first traces of inflammatory irritation in serous membranes, are attended by copious liquid effusion. The more virulently the disease sets in, or the longer it is allowed to maturate, the more turbid does this fluid become, and the more mingled with coagulable substances, until at length fibrinous effusion ensues,—with membranaceous deposition in the serous cavities, and with hepatization in the pulmonary substance. Inflammatory œdema is characterized by this, that the fluid secreted is rarely quite limpid and of aqueous consistency, but rather turbid and viscid, and infused with blood; that the consistency of the pulmonary texture is always more or less diminished; and that pneumonia in one or other of its grades is usually coexistent.

A perilous form, kindred to the inflammatory, is that immediately consequent upon the rapid disappearance of dropsical accumulations elsewhere. This form is sometimes observed in Bright's granular kidney, in heart disease, and even in mere passive œdema of the lower extremities. A marked subsidence of fluid within the subcutaneous cellular tissue, rapidly occurs without any augmentation of any aqueous excretion; the event often proving directly fatal.

The diseases of the *heart,* most liable to produce œdema of the lungs, are those founded either in an impediment to the circulation on the left side of the organ, or else in an acceleration of it on the right side,—in a word, to defects of the valves, and to hypertrophy of the right ventricle. These organic affections invariably engender a certain amount of congestion in the lungs, which, under favorable circumstances, immediately causes serous effusion. *Tumours* pressing upon the pulmonary veins may, in like manner, occasion œdema,—to wit, degenerate bronchial glands and cancerous masses; the latter, especially, where they penetrate into, and clog up the caliber of the vein.

The diseases of the *liver,* which so frequently lead to general dropsy, are not equally productive of infiltration of the lungs. I have, however, repeatedly noticed this condition in granular affection of the *kidneys.* I may here notice a circumstance that has struck me as remarkable; namely, that where in general dropsy which proves fatal, the one lung is found uniformly adherent to the pleura, and the other not, the former is œdematous and the latter compressed by hydrothorax.

That the eighth pair of nerves is deeply concerned in the production of pulmonary œdema is known, partly by direct experiment, partly by certain diseases, partly again by the effect, upon that morbid state, of particular medicinal agents which directly influence the par vagum, as emetic tartar, squill and digitalis. According to Brachet and J. Müller, division of both pneumogastric nerves in the neck, proves fatal, chiefly owing to sero-sanguineous infiltration of the lungs, and accumulation of serum and mucus in the air-passages. In like manner, we see many diseases of the brain, as apoplexy and softening,—or injuries of the head,—terminate fatally amid suffocative catarrh, and afterwards exhibit both considerable hypostasis of the blood within, and an œdematous state of the lung. Even the pulmonary œdema, so frequently developed during the last agony, is probably due to a gradual extinction of the energies of the par vagum.

The condition of the air-passages in pulmonary œdema varies with the causes to which the latter is referrible. The mucous membrane is reddened in active, and blackened in passive œdema, unless in the latter some complication affects the air-passages. The expectoration likewise greatly varies, being sometimes thin and watery, rarely tinged with blood,—sometimes rust-coloured (pneumonic), sometimes again mucous, as in catarrh. Where œdema coexists with paralysis of the lungs, expectoration is commonly wanting, and after death the bronchi and trachea are found loaded with frothy and sero-mucous fluid.

## SECT. III.—HÆMOPTYSIS AND APOPLEXY OF THE LUNGS.

Hemorrhage from the lungs occurs under a great variety of circumstances, being sometimes a primary affection, at other times but a secondary symptom of some other disease. The former case alone concerns us here. A distinction, founded upon the seat and character of the affection, is drawn between simple bronchial hemorrhage (genuine hæmoptysis), effusion of blood into the pulmonary vesicles (apoplexy in a restricted sense), and hemorrhage through rupture of the pulmonary texture (pneumorrhagia). To these three may be annexed that mostly fatal hemorrhage arising from rupture of the larger blood-vessels, during the progress of pulmonary phthisis or gangrene.[1]

*Simple bronchial hemorrhage* leaves no traces after death, beyond a lively reddening of the mucous membrane of the bronchia, and repletion of certain of their ramifications up to the trachea itself, with a bright red, frothy fluid. Sometimes, and especially where the extravasation occurs within the smallest bronchial twigs, the blood is slightly coagulated, without being dark-coloured. The surrounding pulmonary texture there displays a dark red tinge, and appears denser than usual, owing to the loaded state of the closely-packed bronchial twigs, and to the gorged condition of their highly vascular filamentous sheaths. The portion of the lung engaged is mostly œdematous. Where the hemorrhage has been so copious as to occasion anæmia, the substance of the lung has a brick-red, or even a brighter hue.

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[1] This happens only where blood-vessels, whilst yet pervious, are assailed by tubercular or gangrenous destruction—the usual obliteration not having taken place. A rare instance of another kind is described by Carswell (Fasc. Hemorrhage, pl. iii, fig. 5). A branch of the pulmonary artery, adherent to a large bronchial tube, became perforated, in consequence of tubercular ulceration, and fatal hemorrhage through the trachea ensued.

This kind of hæmoptysis is seldom fatal, unless when complicated with other morbid processes. It presents itself in its simplest and least hazardous form, in very plethoric individuals, during the years of puberty, generally ceasing spontaneously when the thoracic organs have become fully developed.[1] There is more ground for alarm, when it occurs as a forerunner of menstruation, or as the reflex of hemorrhoidal ailment, in which cases the attacks often recur to an exhausting amount. Even here, however, it hardly by itself endangers life, however serious its immediate and remote consequences (dropsy, &c.) Examples are indeed known of these bronchial hemorrhages acting vicariously for the menstrual or the regular hemorrhoidal flux, with greater or less violence, periodically, during a space of thirty or forty years, the patients at last succumbing under some other disease. (Compare P. Frank's 'Epitome de curandis hominum Morbis,' tom. ii, § 603.) Under these circumstances there is no real local disease, the lung being merely in a state of congestion, or, more correctly speaking, of hyperæmia. A frequent recurrence of this state, however, engenders various changes, which an experienced observer will readily detect, but which are by no means easy to describe. The texture of the lung is heavier, darker-coloured, more succulent than natural, its loose cellular structure being less distinguishable, on account of the thickening of the septa. The bronchial mucous membrane has become less smooth and transparent, and presents a bluish red tint.

Laennec assumes, and apparently with reason, that in these cases the blood is thrown out upon the surface of the bronchial mucous membrane (and of the pulmonary cells likewise) by simple *diapedesis*, or transudation through the coats of the vessels, and is accordingly derived, for the most part, from the capillary connexions of the bronchial artery and vein. By this latter circumstance simple hæmoptysis is readily distinguished from the two forms next to be described, namely, pulmonary apoplexy and the hæmoptysis so frequent in incipient tubercular phthisis, and therefore, in almost every instance, of evil prognosis. (See the respective chapter.) But simple bronchial hemorrhage may be symptomatic only and independent of organic disease of the lung, where there is a general *scorbutic* condition of the blood. Here, besides the well-known changes in the other parts of the body, marks of imbibition may be detected, with violet coloration of the bronchial mucous membrane, and considerable stagnation of thin liquid blood in the pulmonary texture,—circumstances substantially denoting the peculiar character of the hemorrhage. The bloody tinge of the sputa in certain instances of typhus fever is analogous.

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[1] Amongst the least perilous kinds of bronchial hæmoptysis we have to reckon that noticed by travellers as resulting, on the summit of very high mountains, from diminished atmospheric pressure. The inhabitants of the Alps are said to be particularly subject to hæmoptysis, owing to this cause. (See Michéa, "Notice méd. sur quelques cantons de la Suisse," Gaz. des Hôpit. 1840, No. xli.)

Effusion of blood into the air-cells, or into the general pulmonary texture, with or without its rupture, but with loss of consciousness in the patient, through sudden suspension of the main springs of vital action, constitutes proper *apoplexy* of the lungs. In this sense the term apoplexy is received, since the time of Hippocrates, as applicable to the lungs as well as to the brain; for, although the phenomena may not, in every instance, all occur at the same moment, still there is always, from the very first, a deficiency of vital reaction—a degree of prostration, which forms the chief and most striking characteristic. Death generally ensues very speedily; in rare instances, however, resolution of the apoplectic clot takes place, and a favorable issue has been traced to its inclosure within a membrane, just as in cerebral apoplexy. Pulmonary apoplexy is, for the most part, unaccompanied by hæmoptysis. In 17 fatal cases observed by Louis, hemorrhage from the air-passages only occurred in two instances. Where the effused blood immediately coagulates, the sputa are not bloody; where it does not, always.

In an anatomical point of view, pulmonary apoplexy has been very properly distinguished into that in which the effusion is chiefly thrown out into the air-cells, and into that in which it is produced by rupture of the pulmonary texture. This anatomical division is further borne out by the etiological relations, and by the vital symptoms. In the former variety the changes alluded to occupy either individual circumscribed spots, in the midst of a lobe of lung, or several scattered patches, or finally (though in rare instances, one of which occurred to myself) an entire lobe, if not the greater portion of a whole lung. But wherever the disease has been sudden, and of short duration, the adjacent pulmonary texture is quite healthy; the nearest bronchial twigs, however, are deeply reddened, and the implicated ramifications of the pulmonary artery (Laennec says, of the pulmonary veins) filled with black coagulated blood. The apoplectic portions are rounded off, correspond in form with the lobes, and are from one to four cubic inches in bulk. At these spots the parenchyma is heavy, distended, non-crepitant, consolidated, and of a dark cherry red, a blackish blue, or more frequently a pitch black colour. When cut through, it presents a black surface of nearly uniform aspect, broken only by the larger vessels and bronchial tubes, which appear as whitish cords, and granulated, like the hepatized portions in the second stage of pneumonia, only that *in apoplexy* the granular prominences are firmer and more irregular. Upon the whole, the apoplectic patches, whilst recent, can hardly be said to be softened; but their elasticity is gone, and they become incompressible, fragile, and dry, yielding only a little thick bloody fluid, on being forcibly squeezed, or scraped. Such portions of lung, when exposed to maceration in water, or to the action of the atmosphere, change from black to bright red. Cruveilhier (livr. xxx) asserts that, where this is the case, the pulmonary substance has given way at the centre. I have been unable, however, to demonstrate any dilaceration of the kind; the blood would rather appear to be thrown out from twigs of the pulmonary artery, directly into the pulmonary cells and the smaller bronchial ramifications, and there to become consolidated, without undergoing any further change.[1] The apoplectic centre owes its defined limits precisely to the absence of rupture, the sanguineous effusion being restricted to a system of pulmonary cells dependent upon particular ramifications of vessels, in short, to a single lobule, whose covering separates what is healthy from what is diseased. The notion that the apoplectic clot was formed by the reflux of blood effused into the bronchia, is erroneous.

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[1] Compare G. Gluge (Mikrosc. Untersuchungen zur Pathologie. Heft i, p. 58).

In those cases in which pulmonary apoplexy is associated with spitting of blood, the parts surrounding the extravasation are, as Hope correctly states, found infiltrated with a vast quantity of half-fluid blood, and the bronchial tubes loaded with a sanguineous, frothy fluid, or with coagulated blood. (Gendrin's first grade,—sanguineous infiltration without induration.) The substance of the lung is here always, more or less, of a bright red; for the access of air is not completely and suddenly intercepted, as in the apoplectic parts. These infiltrated surrounding portions are convertible, by ablution and the repeated affusion of water, into seemingly healthy pulmonary texture, whilst, in the apoplectic centres, the blood can only be washed out at the circumference, and that with difficulty.

Where the patient survives the apoplectic seizure for some time, changes take place which lead either to recovery or to further mischief. The earliest change occurs with the blood itself, which, from being simply coagulated, now separates into its solid and its fluid parts. This occasions a softening of the pulmonary substance, and a lighter colouring, verging upon brown, in the implicated portions, which acquire a certain resemblance to red hepatization; at a later period, inflammation may be thence developed. The surrounding pulmonary texture is either gorged with blood, or else œdematous.[1] In tubercular habits, tubercle may become deposited in the very heart of the apoplectic patches, as shown in one instance by Andral (Clin. Méd. 4 ed. t. iv, p. 36.)

Under the most favorable circumstances, the accumulated blood is removed by degrees, and that apparently in a proportionately short space of time, partly by the bronchia, partly by the absorbents. A soft, darkish spot is left, which remains, for a long time, devoid of elasticity and impervious to air. Where resolution does not take place, and the fluid portion of the blood is alone absorbed, whilst the solid is left,—the degenerate lobule shrivels up, hardens, and becomes for ever impermeable. It is not unlikely that certain accumulations of black substance, occurring in other situations within the lungs, besides their apex, are the residue of clots thus reduced. It is not yet decided whether the entire mass ever becomes isolated within a cyst, as in the case of cerebral apoplexy. Bouillaud (Arch. Gén. de Méd., t. xii, p. 399) observed a case in which the effused blood (rupture having probably taken place) became shrouded within a thick, fibrinous, false membrane of a grayish colour, and very like a cyst; the disease was, however, of too short duration for the said membrane to become organized and effect a cure. The whole mass mostly softens down, proving, as an extraneous body, a source of irritation to the surrounding pulmonary substance; inflammation ensues, the diseased patch suppurates, and the effete matter having been discharged through the bronchia, a cavity remains, resembling that produced by abscess or by gangrene. In all these instances the reopening of those branches of the pulmonary artery which have been clogged up with coagulated blood, is an indispensable condition. Where, on the contrary, the obstruction becomes permanent,—and extensive enough to detach the portion of lung concerned from the circulation, gangrene inevitably follows. (See Gangrene.)

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[1] Knox (Edinb. Med. and Surg. Journ. Oct. 1836, p. 404), who submitted lungs in this condition to a minute examination, is of opinion that the aforesaid apoplectic change, attended with secondary softening, is identical with the *soft pulpy tubercle* of Baillie.

The other form of pulmonary apoplexy, where blood is thrown out, not into the air-cells, but into the *cellular texture*, is far less frequent. Here there is always laceration of the pulmonary texture, the blood becomes rapidly diffused, and within a brief space of time may overwhelm an entire lobe, if not the greater portion of a lung. A cavity is thus established, filled, partly with fluid and coagulated blood, partly with the remains of the ruptured, drenched, and utterly degenerate pulmonary substance. Where the blood extravasated is venous, the entire mass is dark red,—almost black; otherwise, it is more of a brick-red colour. The line of demarcation between the diseased and the healthy texture is not so regular nor so sharply drawn as in the preceding form of apoplexy, the confines being, on the contrary, jagged and irregular. (Carswell has delineated this very accurately—Fasc. vi, pl. ii, fig. 5.) Where the extravasation extends to the surface of the lung, the pulmonary pleura may, unless guarded by firm adhesions, give way, and the effusion penetrate into the cavity of the pleura. Several examples of this kind are related by Gendrin,[1] and one figured by Carswell.

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[1] Gendrin (System der prakt. Heilk. Uebers. v. Neubert). Bricheteau (Arch. Gén.,

I am indebted to Professor Radius (of Leipsic) for the opportunity of examining a case of very extensive hemorrhagic rupture of the substance of the lung. In this instance an aneurism of the arch of the aorta, the parietes of which had formed adhesions with the apex of the left lung, had opened and discharged into the substance of the lung, converting the greater portion of the superior lobe into a soft mass, with scarcely a vestige of its natural texture. Within various-sized and irregularly-torn meshes, were seen distinct and separate clots of blood, somewhat dry and grumous, of a pale brown-red colour, and intimately blended, as if mashed up, with filamentous remains of the pulmonary substance. The disorganization was, however, not distinctly circumscribed, but passed gradually into the healthy structure, although in such a manner as, through compression of the lacerated portions of lung, to set limits to the progress of the extravasated blood. In a similar case related by Néret (Arch. Gén. Juin. 1838, p. 203), an aneurism of the left subclavian artery had burst within the apex of the left lung, and there formed a cavity, as big as a child's head, and filled with irregular concentric layers of fibrin. The case proved mortal within a week, amid repeated hemorrhage from the air-passages.

The *causes* of pulmonary apoplexy are extremely various, partaking not merely of a mechanical or dynamical, but probably, also, of a chemical nature. In most instances several cooperate, although we are not thoroughly cognizant of any but the mechanical ones, such as undue afflux of blood to and stagnation in the lung. Accordingly, hypertrophy of the right ventricle of the heart first deserves notice; but although present in most of the examples mentioned, it is to be regarded neither as a necessary, nor even, when met with, as the sole cause. Hope observed it in two thirds of all his cases, and the same proportion holds with respect to those recorded by Cruveilhier, Laennec, and Gendrin.[1] (See Hypertrophy of the Heart.) Defects of the valves on the left side of the heart, either alone or in conjunction with hyper-

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série 2, t. xii, p. 400,) relates a very extraordinary instance of the kind, which terminated fatally within half an hour.

Gendrin appears, however, not to admit the influence of hypertrophy of the heart upon apoplexy. (Gaz. des Hôp. 1840, Mai.)

trophy of the right side, very readily give rise to stagnation within the lesser circulation; accordingly, coarctation of the left auricular orifice, hypertrophy and ossification of the mitral or semilunar valves of the aorta, were of frequent occurrence. A preternatural condition of the pulmonary texture itself appears, however, to be always a predisposing cause, bygone inflammatory disease being, as I can myself attest, in this respect predominant. Aneurismal or varicose (?) dilatations of the larger vessels are, in like manner, enumerated amongst the mechanical agencies. Under the head of dynamical causes may be reckoned violent congestion of the lungs, associated with anormal innervation, as in habitual drunkenness. Hohnbaum,[1] to whom, with Laennec, we are indebted for the first exact investigation of the disease, states that persons of an apoplectic habit, with a thorax of dimensions not proportioned to those of the rest of the frame, are especially prone to apoplexy of the lungs. Finally, a certain chemical change in the mass of fluids, especially in the blood, merit attention in an etiological point of view. Cruveilhier (livrais xxx) simply designates this as scurvy, and adverts particularly to those cases in which hemorrhagic effusion takes place in several organs at once. In drunkards, the anormal quality of the blood certainly favours the production of pulmonary apoplexy. Both sexes appear, at mature age, to be equally susceptible of the disorder.

In conclusion it may be noted, that a cessation of respiratory action, with extreme stagnation of blood, and serous infiltration of the pulmonary texture—in a word, paralysis of the lung—has been erroneously set down as pulmonary apoplexy.[2]

## SECT. IV.—FŒTAL CONDITION OF THE LUNG AFTER BIRTH.—ATELECTASIS.[3]

This disease consists in the imperfect expansion of the lungs by the first inspirations after birth; that is,—in a permanence of the fœtal state, in the lung of the new-born infant. It is to be regarded as a disease dependent upon restricted functional development at the time of birth, and not upon any original defect of formation in the respiratory organs. When at all extensive, atelectasis terminates sooner or later in death; under more favorable circumstances, however, in early and complete, or in tardy and imperfect recovery. For, during the first days after birth, it is still possible that the evil may be surmounted by the vigorous penetration of air; whereas, at a later period, organic changes supervene, which for ever incapacitate the undeveloped portion of lung from performing its proper office. There is, however, still wanting a complete account of atelectasis, as regards both materials and pathological deductions. I shall now endeavour to furnish as faithful an anatomical description as the case will admit of, the result partly of E. Jörg's and partly of my own research.

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[1] Ueber den Lungenschlagfluss. Erlangen, 1811.

[2] See, for example, Rev. Méd. 1837, t. i, p. 370, and several of Hohnbaum's cases.

[3] From the adjective *ἀτελὴς*, imperfect, and the verb *ἐκτείνω*, I draw out. (Ed.)

An entire lung, or even an entire lobe, is seldom found in a state of atelectasis,—but for the most part only single and scattered lobules. Experience shows that certain portions of the lungs are especially prone to retain the fœtal condition, namely, the inferior lobes of both lungs, and the posterior half of the remaining lobes generally. Still examples occur of several lobules near the anterior surface being found in like manner undeveloped.

The diseased patches display a brown-red or rather blueish-red colour, which is more intense if the whole lobule is uniformly unexpanded,—in which case, it is marked off by a sharp contour from the surrounding pale-red healthy substance. Where, on the other hand, scattered cells within such a lobule have become inflated, the violet colour is interrupted here and there, and passes by a gradual transition, and without any distinct boundary, into the natural shade. A distinguishing feature of atelectasis is however this, that the above patches upon the surface of the lungs always exhibit a depression, the superincumbent pleura remaining perfectly smooth and polished. A lobe either entirely, or for the most part, in this condition, is never found enlarged, but on the contrary, of much smaller dimensions than the others, and almost as collapsed as in the fœtus; in general deeply imbedded within the thorax, and drawn towards the entrance of the bronchi and blood-vessels. Hence single diseased lobules do not attain the same elevation of surface as the healthy ones with which they are surrounded, but, as already stated, form depressions more or less considerable, so that the general aspect may be likened to the dimples created by emphysema, in adult lungs. Neither by incision nor pressure is any crepitation produced, unless where a few air-cells happen here and there to have become expanded. The same delicate reddish froth is never found here as in the healthy parts of the lung, but merely a small quantity of serous, slightly sanguineous fluid. The cut surface appears smooth,—uniform,—without a vestige of granular elevations. The whole of the diseased structure is not softened, but rather of a hard character, still without the tenacity of the healthy parts. When a patch so situate is cut off and placed in water, it sinks to the bottom.

When atelectatic infants die a day or two after birth, it is generally possible to dilate, artificially, the undeveloped parts. The depressed lobule is then seen to rise by degrees to the level of the rest, and to assume the colour, permeability, and other characters of sound lung. Up to this point, had other circumstances been favorable, perfect recovery might have taken place. Where, however, the little patients have survived for weeks or months, this inflation seldom succeeds, or only imperfectly. At this juncture the unexpanded pulmonary cells are for the most part coherent: a remarkable fact, seeing how long the lungs continue unexpanded in the fœtus, without adhesion ever taking place. What ulterior transformations go on in the diseased parts, it is not yet satisfactorily determined; it is, however, more than probable that not a few indurations and depressions, especially the small calcareous concretions, sometimes occurring without any obivous cause, at particular spots within the lungs, (generally at the back of the inferior lobes,) are referrible in some measure to the above source. At all events, it may be observed generally that, in atelectasis, the boundary line between the diseased and the healthy substance becomes less and less distinct, in proportion as life is prolonged. At the earliest period of infantile existence the contrast is very decided, the diseased parts being immediately surrounded by pulmonary texture in all respects natural and healthy.

In infants who had died of atelectasis E. Jörg invariably found the foramen ovale of the heart unclosed; a fact confirmed by myself, but which, at that age, is not unusual. The brain was in a congested state. When death followed shortly after birth, the body had the appearance of being generally well developed, but was extensively ecchymosed; the hands and toes were clenched; and there was foam in front of the nostrils and of the closed mouth. Where, however, the disease had been of some standing, the body was wasted and the skin loose and wrinkled. Jörg remarks, that under these circumstances, both the affected and the adjacent parts are found inflamed, if not in a state of suppuration. The details of Jörg's cases, however, clearly show them to have been complicated. Inflammation is neither necessarily nor even frequently the sequel of atelectasis, for often as I have witnessed this disease I have never met with a case of inflammation which could be directly traced to the diseased lobule. I have even seen a case of genuine pneumonia with hepatization of the inferior lobe, one portion of which being *atelectatic*, had not participated in the inflammation, but presented hard, knotty, depressed patches in the midst of expanded, softened, hepatized substance.[1] Lobules retaining the fœtal condition, are quite passive in relation to other morbid processes, and especially to inflammation; the examples given by Jörg appear rather to have been cases of real pneumonia.

It is here requisite to state that *atelectasis*, with the characteristic features above described, is not always accurately distinguished from other diseases; that condition having, in some instances, not been duly recognized, and, in many more, been confounded with quite dissimilar pulmonary affections. Indeed, the greatest discrepancy of opinion, or rather the greatest confusion prevails, so that I am here under the necessity, however hazardous it may appear, of submitting those discordant views to the ordeal of a critical examination. The French writers on children's diseases, who first turned their attention to the changes in the pulmonary texture of new-born infants, refer *atelectasis* chiefly, if not exclusively, to the head of *pneumonie lobulaire*. Several German authors have, without further inquiry, adopted this view, (Vernon, Der Arzt am

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[1] The second case of Valleix (Cliniques des Maladies des Enfans nouveau-nés, 1838,) offers many points of resemblance with the above.

Krankenbette der Kinder, Wien, 1838, vol. ii, p. 54, &c.;) while others, admitting the peculiarities of atelectasis when associated with ordinary pneumonia, continue nevertheless to connect it, either directly or indirectly, with the same inflammatory process. It will not be difficult to show wherein lies the essential difference between the two. In atelectasis, the colouring of the diseased portions of lung always approaches more to a violet, their exterior appearing smooth and glistening, so as to contrast with the dull, brown-red surface of inflammation. In inflammation, again, the diseased portions are preternaturally distended, whilst in atelectasis they are collapsed, and inferior even to the healthy texture in volume,—but susceptible, provided the disease has not lasted too long, of artificial inflation, and capable, through its means, of acquiring a perfectly natural appearance. In inflammation, the pulmonary texture is softened, in atelectasis it is hard, and the cut surface is not granular, but smooth. Where no complication exists, the anatomical characters of a first or third stage of pneumonia are not discoverable either in or near the diseased patch; in short, we have nothing like pneumonia except the solid, non-crepitant mass, which has been confounded with the second stage of that disease, namely, with red hepatization. Where single pulmonary cells have been found dilated in the midst of an undeveloped lobule, the absence of softening, and of the peculiar, congested, humid character of its texture, offers a wide difference between it and the first stage of pneumonia. A portion of lung retaining its fœtal condition allows a little thin, dark, apparently natural blood to escape upon pressure: in the first degree of pneumonia a tolerable quantity of a turbid, bloody fluid, mingled with fibrin, and with a few minute air-vesicles,—in red hepatization, a tenacious dirty-brown reddish,—in gray hepatization, a large proportion of grayish-yellow purulent fluid may be expressed. Atelectasis usually affects both lungs,—pneumonia is, for the most part, confined to one. Finally, the secondary phenomena attendant upon pneumonia, as inflammation of the pleura and of the bronchial mucous membrane, softening of the bronchial glands, fibrinous concrements within the heart's cavities, &c., are wanting in atelectasis. But the peculiar characters of this fœtal condition of the lung are only thus marked during the first few weeks after birth: subsequently, when, as already stated, ulterior changes take place, it becomes extremely difficult to form an exact diagnosis from mere cadaveric inspection.

Jörg observed atelectasis in children, in whom the first act of breathing had been imperfectly accomplished, either because they were puny and feeble, or because they had been hurried into the world before placental respiration had been altogether suspended, and the necessity for pulmonary respiration become sufficiently potent to stimulate all the muscles of inspiration. He therefore concluded, that it was due to the inhaled air not sufficing to effect complete expansion of the lungs;[1] a view corroborated both by the symptoms and course of the affection. This partial introduction of air might be deemed at variance with the physical laws of respiration, inasmuch as the atmospheric pressure must necessarily distend the entire lung equably, not to the exclusion of a lobe, and, still less, to that of a lobule. The objection, however, falls to the ground, when it is considered that the operation of these laws is the result of previous muscular action. Moreover, there is a great analogy between the pathological relations of primal respiration and those of many other affections,—pleurisy, for instance, (see that article,) where the one half of the chest—and especially in partial pleurisy, where certain portions—do not at all share in the movements of the remainder,—and where, again, after the absorption of circumscribed empyema, those very portions collapse and become totally inert, a partial deformity of the chest being the well-known result. We need, therefore, be at no loss to understand how defective breathing may originate in a merely partial activity of the intercostal or other respiratory muscles. It ought to be added, that such portions of the lungs as commonly require several forcible inspirations for their due expansion, are especially prone to remain in the fœtal condition. To conclude, it can hardly appear singular that atelectasis should be generally more extensive in the right lung, when we call to mind how the capacity of that half of the thorax is diminished by the great size of the liver in the fœtal state.

Atelectasis has been confounded with pneumonia by most writers on the diseases of children, as their own publications amply prove; for we find specified, under "infantile pneumonia," so much that is peculiar and enigmatical,—so many deviations from the same disease in the adult, as to render it obvious that two maladies of quite a dissimilar nature are brought under one rubric. Even the account they give of the appearances after death, are in many instances conclusive as to the justness of the above assertion; and that without reckoning the previous symptomatic relations,—namely, the frequent absence of true inflammatory phenomena, the want, or irregular accession of fever, and the very slender information derived from percussion and auscultation.[1] The existing difficulties have induced several writers to assume a double form of infantile pneumonia. Rilliet and Barthez [2] distinguish an acute and a chronic type: the first occurs in children, somewhat advanced, commencing with the symptoms of catarrh, and resembling in general the pneumonia of adults; the chronic form, on the other hand, assails for the most part new-born infants, and agrees, in symptomatic and anatomical characters, very closely with atelectasis. Thus Billard [3] affirms congestion to be more common in infancy,—true hepatization at a more advanced period; and Heyfelder (l. c. p. 140) confirms the remark made at the hospital for sick children at Paris, namely, that in children beyond the sixth year, pneumonia bears a close affinity to that in the adult. But this division of cases does not remove the difficulty; because, as Gerhard has shown, atelectasis chiefly concerns infants not above twelve months old.[4] Cruse,[5] who very properly rejects the idea of atelectasis being a pneumonia or bronchio-pneumonia, met with genuine inflammation of the lungs in young infants very rarely, and found the after-death appearances to bear a near resemblance to those of the disease in adults. His assertion, however, that in quite young infants this inflammation assumes only exceptionally, if ever, the same form as in adults, is in contradiction with experience,—so far at least as the anatomical characters are concerned. Again, the views promulgated with reference to the organic changes affecting the pulmonary texture, further evidence the obscurity and confusion that prevail. Valleix in his otherwise highly valuable work (p. 195), on the one hand, declares the pneumonia of children (though confounding it with atelectasis) to be identical with that of adults; whilst on the other he is perplexed by certain incongruities, really belonging to atelectasis. His greatest difficulty is, to account for the *"hepatized"* patches being always hardened, instead of softened, and for their cut surface being smooth, instead of granular. In the new-born child he never met with those secondary results of pneumonia, so constant in adults, and whatever has been encountered of that nature by others, he is forced to ascribe to some other morbific source. He also pointedly adverts to his own experience, as well as to that of others, touching the rarity of pleuritic or bronchitic complication. Billard (l. c. p. 534) expressly seeks to show that this disease essentially differs from the pneumonia of adults. He says: "the pneumonia of the new-born obviously arises from stasis of the blood in the lungs, the stagnant blood operating as a foreign body," &c.; and again, "The cause of the inflammation is purely mechanical, and it is not to be wondered at that the pneumonia is very circumscribed, and indeed limited to the patches originally affected."[1] Seifert (Die Bronchial-pneumonie der Neugeborenen, &c., 1837) in a like manner compares the state of the lungs in question with hypostasis, (where, however, softening takes place, and not hardening,) the inflammation being rather of a congestive, venous character. He assumes four grades of bronchio-pneumonia (p. 94); these do not, however, represent the same inflammatory disease in different degrees of advancement, but different forms of one and the same condition, the description of which cannot fail to recall atelectasis to mind. Cruse (l. c. p. 113) endeavours to convince him that neither hypostasis nor inflammation is present, and hints at the possibility of atelectasis; but, without pursuing the subject any farther, refers the pathological changes to bronchitis. Rilliet and Barthez come nearest to the truth, in describing under the pneumonia of the new-born a peculiar alteration, namely, carnification. Here the affected portion is commonly situate at the base of the lung; it presents a smooth, compact cut surface, which, on pressure, emits a thin sanguineous fluid; it is sometimes associated with hepatization, and then the lung resembles that of a fœtus that has not yet breathed. The manner in which the disease spreads within the lungs is opposed to the course of pneumonia, but consistent with that of *atelectasis*. Seifert generally found both lungs similarly affected, and Valleix mentions, from researches made by Vernois at the foundling hospital of Paris, that, out of 113 cases, there were 100 in which both lungs were simultaneously diseased.

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[1] This explanation was given by J. C. G. Jörg (Professor of Midwifery at Leipsic,) in 1831, and published by his son, E. Jörg, M.D., the year following. See his Dissertatio de pulmonum vitio organico, &c. Lips. 1832; and Die Fœtuslunge im geborenen Kinde, &c. 1835.

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[1] Compare Heyfelder (Studien im Gebiete der Heilwissenschaft, vol. ii, p. 136,)—Kluge (Vereinszeitung, 1835, No. xxx.)

[2] Maladie des Enfans. Prem. partie, 1838.

[3] Traité des Maladies des Enfans, nouv.-nés. 1833.

[4] Journ. des Connaiss. Médico-Chir. Sept. 1835.

[5] Ueber die acute Bronchitis der Kinder, p. 118. 1839.

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[1] Cruveilhier appears to entertain similar opinions; although, where death occurs within a day or two after birth, he believes that the process of infiltration and inflammation must have commenced *in utero*. Out of twelve of his cases several, and of his figures Nos. 1 and 4 (livr. xv, pl. 2,) belong to *atelectasis*. He expresses himself thus cautiously concerning the disease: "Il meurt autant d'enfants nouveau-nés que d'adultes par les poumons."

Unequivocal cases of infantile pneumonia, whether lobar or lobular, such as I have myself examined, and as Kiwisch has published (Oesterreich. Medic. Jahrb. N. F. vol. xxi, Stück 4, p. 534,) afford, on the other hand, the strongest negative grounds for establishing atelectasis as a distinct form. In the great majority of cases, Kiwisch found but one lung affected, the hepatized patches being invariably softened, and rendered cognizable from without, by a dull grayish colouring; conjointly with hepatization the other stages of pneumonia were present, including that of abscess; in every case there was pleuritic effusion more or less copious; bronchitis was seldom noticed,—endocarditis in one case; finally, percussion and auscultation always afforded the ordinary results, whilst the asphyctic and cyanotic phenomena were comparatively trivial.

In concluding these critical remarks, I consider myself entitled to draw the following inferences: New-born infants are prone to an organic affection of the lungs, altogether distinct from pneumonia, and dependent upon imperfect inspiration after birth, by many pathologists confounded with pneumonia, and by Rilliet and Barthez designated as carnification. The greater number of cases of pulmonary disease occurring at the earliest period of infantile life, and set down as pneumonia, may be looked upon as cases of atelectasis. The last assertion is, however, to be taken with some reserve,—inasmuch as, in vast lying-in or foundling hospitals (Kiwisch), pneumonia is apt to become epidemic with new-born infants, and, under these circumstances, to attain a numerical preponderance over *atelectasis*.

# CHAPTER IV.

## DISEASE OF THE AIR-PASSAGES.

### SECT. I.—PULMONARY CATARRH GENERALLY CONSIDERED.

This affection, peculiar to the mucous membranes, and in all its forms most frequent, by far, in the mucous membrane of the respiratory organs, is of vast import, owing to its serious consequences, its various terminations, and its multiform complications. Vestiges of its existence meet the eye of the attentive observer in almost every instance of cadaveric examination. With a view to render matters more easily intelligible, attempts have repeatedly been made to classify the disease according to its locality. Very marked distinctions, however, in this respect, are not borne out by facts; it seems expedient, therefore, to begin by discussing the subject generally, in order to get rid of some of the confusion in which it is involved.

Simple catarrh assails alike particular patches, or the whole expanse of the mucous membrane of the air-passages, though seldom with uniform intensity, or at one and the same moment. In most instances, its progress is from without inwards; thus from the larynx it will advance, sometimes with incredible rapidity, along the mucous membrane of the trachea and bronchia. More rarely, it invades a distinct division of the bronchial ramifications, spreading from thence upwards, into and beyond the trachea. This latter event is wont to occur more particularly where the bronchial mucous membrane, or else the pulmonary texture, has undergone changes, connected or not, with bygone catarrhal states. Acute catarrh may stop short at any one of its stages, or pass from thence into the different varieties of chronic catarrh, and there terminate without any ulterior mischief. It even happens that, whilst in one patch of the bronchial mucous membrane the catarrhal irritation arrives at its natural termination, a second patch degenerates, at one stage or another, into a chronic state. A further question arises, namely, whether, as taught by writers on general pathology, catarrh ever terminates without expectoration. Such might certainly *appear* to be the case: but a reference to the microscopic researches to be hereafter specified, will show that no catarrh, however slight, is exempt from that detachment and regeneration of epithelium layers, which, when copious, used to be designated as a critical secretion of mucus,—as mature sputa,—but which, when scanty, may possibly escape ordinary observation.

One attack of catarrh leads under favorable circumstances to another. Again, chronic catarrh is increased, both in extent and virulence, by the supervening of a fresh acute attack; and such frequent revival of the disease tends materially to impede recovery. To this it is to be added that protracted catarrh brings about certain changes of structure, which in like manner operate as exciting or sustaining causes of the original affection, at the same time implicating other organs, and exhausting that innervation of the respiratory apparatus which is essential to life.

But apart from any such pathological sequence, catarrhal irritation may pass directly into intense inflammation, and thus assume a serious and even perilous aspect. It would be difficult to determine the precise moment when such passage, from genuine catarrh to inflammation, takes place, the two being closely allied to each other, and by many pathologists regarded as mere gradations of one and the same affection. In a strictly anatomical point of view the characteristic difference between catarrh and inflammation is not well defined, and can only be determined by reference to the vital phenomena; suffice it to say, that the various forms of inflammation of the air-passages are, in the majority of cases, developed from catarrhal affections. For this reason it might appear expedient to bring all these diseases under the general head of catarrh. We must, however, on the other hand, admit that inflammation of the air-passages may arise from causes wholly independent of catarrh.

Inflammation resulting from violent augmentation of catarrh, is characterized, not merely by the secretion of pus from the diseased surface, (by the microscope, undoubted pus-globules have been detected in the secretions of ordinary catarrh,) but by the formation of a plastic inflammatory product, as also by implication of the textures subjacent to the mucous membrane. It must, however, not be forgotten, that through the superficial extension of the catarrhal process, the affection may assume an inflammatory character at some particular spot, without the limits of catarrhal irritation being elsewhere overstepped.

It is highly probable that catarrh exercises over the nerves some pathological influence productive of both primary and secondary disturbance to their function. In what manner this occurs is not very clear. The fact did not, however, fail to strike even the older pathologists, that catarrh, in itself slight, sometimes superinduces sudden paroxysms of spasm or paralysis, which threaten immediate suffocation. In children, this manifests itself as spasm of the glottis, or crowing inspiration, with or without hypertrophy of the thymus gland, and independently or not of swelling of the lymphatic glands along the course of the trachea or around the bronchia. In croup, the remissions and exacerbations are likewise sufficiently striking; but the nervous complication of catarrh is most conspicuous in hooping-cough. Adults are little prone to nervous affections of the kind, if we except that aphonia, sometimes the attendant, sometimes the long-continued sequence of catarrh, and unconnected with any obvious disease of the vocal chords. In more advanced years, catarrh frequently predisposes to purely nervous asthma; and, in old age, sudden and fatal palsy of the lung is not uncommon during a simple catarrhal affection.

There are, besides, other complications, proceeding either from some constitutional cause, or else from the disease itself, of which they are the organic and almost necessary sequelæ. Thus scrofula will invest chronic catarrh with the character of an obstinate bronchial blennorrhœa, and eventually, perhaps, lead to tubercular disease of the respiratory organs. The same thing applies to gout, which, through repeated attacks of catarrh, is made to settle, as it were, upon the bronchial system, imparting a peculiar character to its augmented secretions, furthering the development of secondary organic mischief, and frequently inducing palsy. On the other hand, protracted catarrh sooner or later determines atrophy of the pulmonary substance, deviations with respect to the space occupied by the heart, a cyanotic state, and, finally, dropsical collections. Hence it follows that catarrh, however trivial its commencement and imperceptible its progress, is apt to become a serious annoyance during life, and even in one way or another to cause death.

We shall now proceed to describe acute and chronic catarrh, and the transition into inflammation; under which last head, œdema of the glottis, croup, catarrhal pneumonia (pneumonia notha), and inflammation of the textures subjacent to the mucous membrane, will be specially considered. This will be followed by an account of the organic results of catarrhal disease, namely, hypertrophy of the mucous glands, and of the mucous membrane itself, together with the formation of diverticula and of polypous growths,—dilatation of the bronchial tubes and pulmonary cells, with atrophy of the pulmonary texture generally.

## SECT. II.—ACUTE AND CHRONIC CATARRH.

Catarrh is a disease of every-day occurrence, and spares neither sex nor age. It is fraught with but little danger, unless where widely diffused over the mucous membrane, or seated in the majority of the minute bronchial ramifications, or, finally, where the individual is greatly deficient in vital reaction, either by nature, or from disease.

Acute catarrhal irritation of the respiratory mucous membrane is characterized, at the commencement, by irregular rosy patches, nowhere distinctly circumscribed, but as if shaded off at their circumference. This reddening is occasioned by fulness of the extremely delicate, superficial vessels, which, however, even in this condition, still admit of artificial injection,—according to Gendrin. In a short time, however, the above patches exhibit scattered vermilion points, whilst the pale red vascular network becomes darker, and appears to go to a greater depth. The vermilion specks presently become more numerous, unite to irregular undulating streaks, and ultimately coalesce in patches over the entire surface, imparting to the mucous membrane an uniform tinge, and changing its thin, smooth aspect into one resembling plush. A ramifying of blood-vessels is now no longer visible upon the surface, though very evident in the submucous tissue. The upper layer of mucous membrane is mostly dry, double its natural thickness, and easily torn.

At first the membranous surface is but slightly suffused with mucus; after a while it becomes more dry than natural, but ere long secretes a thin watery fluid, which becomes viscid in proportion as the irritation increases, and soon begins to grow turbid. This turbidness first shows itself in the form of grayish points and streaks in the sputa, which by degrees are rendered wholly opaque. Eventually, there ensues a very copious secretion of thick, yellowish mucus, which, under the continuance of inflammatory irritation, is liable to take on a decidedly purulent character. Pus is apt thus to form in great abundance, sometimes without any perceptible loss of substance in the mucous membrane, which continues to present the appearance already described. In other instances, however, where the catarrhal irritation is developed with great rapidity and intenseness,—the mucous membrane, especially in the upper half of the larynx, the posterior surface of the trachea, and the two main bronchi, exhibits more or fewer distinct patches of superficial erosion. In rarer instances the erosion is more limited, penetrating deeper, and displaying a brown-red base, circumscribed by a whitish linear margin, in whose vicinity a capilliform plexus of vessels is often conspicuous. These erosions are nearly circular in shape, rarely exceed four or five lines in diameter, and are not larger than a lentil. They appear to be the result of predominant affection of the little mucous glands, around whose ducts the mucous membrane becomes superficially softened and abraded. These mucous glands are occasionally found in the shape of whitish or yellowish granules, as big as mustard or millet seeds, around the ventricles of Morgagni (or Galen),—and between the rings, and on the posterior wall of the trachea, where their complete softening and liquefaction may give rise to little isolated ulcers. In this form their distinction from other diseases is extremely difficult, and must depend upon the simultaneous occurrence of other changes. Thus the damage consequent upon *tubercular* disease of the lungs (more minutely described elsewhere), is in part analogous, in part identical with that just noticed, although, within the larynx and trachea, genuine tubercular ulceration and destruction have a very peculiar and characteristic appearance.

Again, in certain cases of *typhus* fever, superficial erosions occur upon the lower surface of the larynx, and above the rima glottidis, besides others more profound, essentially differing from the catarrhal. The erosions of typhus are, at least in the cases seen by me, less prone to spread superficially, and always display a dirtier, and more of a violet hue, than those of simple catarrh.

In *aphthæ* and *diphtheritis,* it is not uncommon for the detachment of epithelium, and the plastic effusion from the fauces, to extend to the glottis, and from thence to the larynx; the true connexion of the disease will, however, in such cases, be easily detected, a remark which applies more obviously to *variolous* affection of the air-passages and to *syphilitic* destruction.

The aforesaid catarrhal changes, so far as the respiratory mucous membrane is concerned, are the result of irritation of the submucous tissue, whereby, as the microscope shows, the metamorphosis of the epithelium layers is disturbed in a remarkable degree. The examination of diseased mucous membranes by the microscope, has of late years led to very interesting results,[1] both as regards acute and chronic catarrh, and their complications with inflammation. I shall, therefore, introduce here some of the more important observations on this head. Henle has shown, in his valuable treatise upon the *epithelium,* that it exists in a peculiar form on the mucous membrane of the repiratory passages, and affords a means of ascertaining the minuter changes of structure in catarrh. It is well known that, in the healthy state, the epithelial investment is being continually, though imperceptibly, cast off,—and regenerated out of the secretion proceeding from the true surface of the mucous membrane. Accordingly, the ordinary natural mucus of the air-passages consists of these extruded epithelium-cells, together with liquid mucus from the muciparous glands. Whilst, however, the epidermis and the tesselated or plaster epithelium[1] consist of various layers of epithelium-cells, in a more or less advanced state of development from without inwards; the epithelium of the air-passages consists of but a single thin layer, which is generated by a dense array of ciliary cylinders. The changes produced by catarrh consist essentially in the following. Between the true surface of the mucous membrane and the epithelium an effusion takes place, at first almost exclusively of serum, by which the epithelial covering is raised, and here and there torn,—allowing the escape of watery effusion. After some time, small isolated groups of ciliary cylinders become separated, and, ultimately, the whole coating either removed all at once, or dissolved, rolled into pellets, and thus rejected. Where the irritation is evanescent, and a new epithelium lining able to form, previously to the old one being completely separated, the disease is brought to a conclusion. Generally, however, the morbific causes continue for a more lengthened period to influence, if not the whole, at least some portion of the mucous surface, rendering the formative process too tumultuous for the generation of normal products. The serous exudation now becomes blended with little irregular, granular bodies (inflammatory spheres), or with regular globules containing a nucleus, which is brought to light by acetic acid (mucus-corpuscles and simple exudation-cells), and soon after with true pus-globules, which are easily recognized by their uniform size, their semi-opaque and delicate granular appearance, and by from two to four nuclei becoming visible on the application of acetic acid. The mucous surface thus furnishes pus without any ulceration. This gradual transformation of mucus-corpuscles into pus-globules, or rather the succeeding of purulent to mucous secretion from the same secreting surface, was first distinctly shown by Vogel, and figured by him in his 8th plate.[1] Henle confesses his inability to discriminate between mucus of morbid secretion, and pus,—but maintains that the former varies in composition, so as to contain epithelium-cells, mature and immature mucus-corpuscles and pus-globules, either together or in succession,—one form passing imperceptibly into the other. Even Gluge, who professes to discriminate between mucus-globules and pus-globules, assumes three forms of catarrhal secretion from the mucous membrane: namely, 1st, mucus-globules, mingled in various proportions with mucous liquor, (mucous catarrh); 2dly, this latter associated with a fibrinous exudation, with which the bronchial ramifications become clogged, (exudative catarrh and also croup); and finally, mucus, more or less commixed with pus, (purulent catarrh).

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[1] See Vogel (Ueber Eiter. u. s. w. 1838, pp. 88, 143;) G. Gluge (Anatomisch. mikroskop. Untersuch. u. s. w. 1838, p. 62, and elsewhere;) Henle (Ueber Schleim u. Eiterbildung und ihr Verhältniss zur Oberhaut, 1838, separately published from Hufeland's Journal). These researches were all made about the same time, and, though they do not altogether agree with each other, they at any rate afford similar results, with reference to the main points.

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[1] [Consult Mr. Paget's 'Report,' in the British and Foreign Medical Review for 1842, p. 264.—Ed.]

The transformation of mucous, into purulent secretion is very rapid; Vogel believes that it may take place within a very few hours, and the secretion return, with corresponding rapidity, to its original standard. But as the irritation is seldom equably diffused over any one portion of the mucous membrane, the effused secretion usually contains, simultaneously, normal epithelium-cells, true pus-globules, and every intermediate gradation between pus- and mucus-corpuscles. In simple coryza, Henle detected, in the secretion from the Schneiderian membrane, cells of every grade of development of epithelium, and mucus-granules of every form, up to what he would consider equivalent to pus-globules. In a female, in whom "suffocative catarrh" (probably bronchitis) had proved rapidly fatal, he found the bronchial mucous membrane of a vivid red, and overspread with a yellowish, easily separable, thin, flocculent membrane. This was made up of coagulate fibrin, of perfectly formed ciliary epithelium, of epithelium-corpuscles,—apparently belonging to a deeper layer, engaged in the act of transition into true ciliary cylinders,—and of globules resembling mucus-granules, but which, on being treated with acetic acid, behaved like pus-globules. The bronchial mucous membrane presented no vestige of epithelium, except a few of the cells last described. Henle concludes that, at least in some catarrhal diseases, the original healthy epithelium does not undergo any change, nor the exudation, with the characteristic mucus-granules, form upon the surface of the existing epithelium, but that the latter is thrown off unchanged, the pathological cells becoming developed underneath, and upon the immediate and denuded surface of, the mucous membrane. It has been before shown that the shedding of the epithelial layer is not a preliminary, but a somewhat later operation. The more intense the inflammatory irritation,—the more numerous the pus-globules,—the slower will be the restoration to the natural condition, the mucous membrane long continuing deprived of the power of generating true ciliary epithelium.[1] A tenacious, homogeneous fluid, with traces of fibre, then shows itself in the expectoration, often for weeks. It is mixed up with scattered nuclei, and little arborescent fibres,—with a multitude of mucus-corpuscles, with imperfect epithelium-cells, and, finally, with pus-globules,—being relatively more or less limpid or opaque. The application of acetic acid renders all these elements of expectoration severally distinguishable.

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[1] [So far as the microscopic characters are concerned, this assertion is correct; the fact, however, of the secretion from a diseased mucous membrane gradually changing from mucous to purulent, independently of ulceration, was pointed out by the late Professor Charles Badham, in 1808.—Ed.]

From the foregoing much may be deduced, tending to illustrate some of the obscurer points connected with catarrh, and its passage into inflammation. We learn, first of all, why it is that the nature of catarrh cannot be altogether determined from mere anatomical data, which would simply indicate catarrh to be a lower degree of inflammation. We further learn why the prognosis must regulate itself according to the particular seat of the catarrhal irritation. For, independently of the influence of the ciliary organs in carrying along the secretion of the respiratory mucous membrane,—it is obvious that a catarrh limited to the larynx, trachea, and larger bronchial tubes, is fraught with but little jeopardy,—inasmuch as the expulsion of the sputa is then mainly accomplished by the impulse of the rapidly expired column of air in the act of coughing, and as there is little risk of obstruction or suffocation, owing to the greater width of the air-passages in the above situations. Where, on the contrary, the catarrhal irritation has assailed the greater portion of the smallest bronchial twigs of one or both lungs, and stripped off the ciliary epithelium, there is far less likelihood of the air-passages being freed from the hinderance to adequate ventilation. When, however, catarrhal irritation is aggravated into inflammation, the secretion is rendered more viscous by the admixture of fibrin, and the fibrous coat of the bronchial tubes (akin to muscular fibre) is paralysed, as Stokes has shown, by the effect of the adjacent inflammation. We cannot marvel therefore that bronchitis should be so serious a disease, associated with such urgent dyspnœa, and with so many signs of impeded transmutation of the blood. On the same ground, it may be readily explained why limited lobular hepatizations, when originating in catarrh, prove more hazardous and fatal than ordinary pneumonia occupying much ampler space.

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[1] In the exudation from the mucous membrane, J. Vogel's so-called granule-cells occur very rarely, and I have only detected them in one particular form of sputa, wherein are seen individual groups of pale, rust-coloured specks, occasioned by granule-cells.

The cure of catarrh, and its passage into the chronic state, are readily explained by the results of microscopic observation, so far as it has gone;—still, positive demonstration is wanting. Thus, whilst with the gradual decline of catarrhal irritation, a better regulated secretive action of the mucous membrane is set up, the epithelium-cells, probably formed in a less hurried manner, reach the surface in a more perfect condition, and ultimately constitute a complete and connected ciliary investment. Where, on the other hand, the disease becomes chronic, the mucous membrane may be assumed to have had its organic function too severely impaired presently to regain the capacity of secreting normal epithelium-cells. Thus denuded of its natural protecting tunic, it will not only go on profusely secreting mucus- and pus-globules, but will, at the same time, be more subject to a repetition of morbific influences, and more prone to a renewal of the catarrh. The fluctuating character of the expectoration, in most instances of the disease, favours such an explanation.

We subjoin a few remarks, touching the varieties of simple catarrh. Of these, *hooping-cough* is by far the most important, in a practical point of view, on account both of its frequency and of the peculiarity of its symptoms and course. All the endeavours of pathological anatomists to refer it to some fixed seat have failed. It cannot be traced, in a satisfactory way, either to a peculiar organic change of the respiratory organ itself, or to affection of the stomach, pneumogastric nerve, or solar plexus. Experience would, indeed, rather lead to the conclusion of hooping-cough being nothing more than chronic catarrh, which, in persons prone to strong nervous reaction,—like children, and equally excitable adults, especially of the female sex,—provokes the well-known paroxysms. It is not fatal in itself, but only through the complications that beset ordinary catarrh. Of all forms, hooping-cough appears most liable to engender emphysema of the lung.

Of *influenza,* again, there is nothing to be said but what applies equally to common acute catarrh. Like all epidemic diseases it assails with marked intensity the general organism, and is particularly prone to merge in the more perilous forms of inflammatory complication. (See Catarrhal Pneumonia.)

*Suffocative cough* has not even in a pathological, still less in an anatomical sense, any claim to be distinguished as a particular species. It constitutes the closing scene to almost every lung or heart affection, and is the result of paralysis depriving the respiratory organ of the power to expectorate the masses of accumulated secretion.

It may here be proper to observe, that every accumulation of mucus found within the bronchia after death, is not due to either simple or inflammatory catarrh. Thus in portions of lung long compressed by pleuritic effusion, the smaller bronchial twigs are almost always found replete with a thin whitish mucus. A careful inspection will, however, show the mucous membrane to be thoroughly sound in all respects. The mucus must, therefore, be regarded as the natural secretion within an inert lobe of lung, withheld from access of air. It is more difficult to account for the same phenomenon in typhus fever. Here we meet with no organic alterations of the respiratory mucous membrane, analogous to the catarrhal or inflammatory (complications excepted), unless in the situation before specified,—namely, the larynx, especially above, rarely below the rima glottidis; secondly, we find the accumulations of mucus in the bronchia of the posterior, inferior portions of the lung, only precisely where we encounter stagnation of blood and infiltration of the pulmonary texture with the fluid parts of the blood; thirdly, the same condition is present in almost equal degree in both lungs; fourthly, the pale violet, or progressively more dingy hue, together with the softened state of the mucous membrane, (which in simple cases I have never found tumefied or thickened,) is seemingly quite identical with the softening and imbibition to which so many other organs are subject, during the progress of typhus.

*Chronic catarrh* requires very brief consideration, inasmuch as there is no characteristic sign whereby, after death, it can be distinguished from acute catarrh. As a general rule, chronic catarrhal irritation is connected with a darker tinge of the mucous membrane, which sometimes inclines to a brown-red, but for the most part displays a violet shade, and frequently presents a dense network of more or less delicate ramifications of vessels. The uppermost stratum of the mucous membrane is, withal, less tumefied and friable than hardened and thickened, as if more intimately united with the deeper-seated layers. The mucous secretion, which, during the first period of acute catarrh is always very tenacious and pellucid, here constitutes a thick, turbid, and for the most part copious fluid. Chronic catarrh, when of long standing, is invariably productive of one or other of the sequelæ, to be hereafter described. It should, however, be stated, that it is usual to class almost every species of abiding irritation and of chronic inflammation of the respiratory mucous membrane, whatever their source, under the general denomination of chronic catarrh. This is done with all the *semblance* of propriety, because neither the signs during life, nor the changes visible in the mucous membrane after death, establish an obvious difference; and again, because a catarrhal complication is really frequent under those circumstances. Thus bronchial irritation, caused by tubercle or by other heterologous growths within the texture of the lung, by disease of the bronchial glands, and, finally, by most heart affections, is termed, alike, chronic catarrh, and it is hardly necessary to add how greatly this want of due discrimination tends to embarrass the subject. *Genuine chronic catarrh* invariably arises out of an acute state. It is very common in children, with whom, especially if they be of a strumous habit, it may abide for several years, ceasing only at the age of puberty.

In the bloom of life catarrh seldom becomes chronic, especially in the male sex, and for the most part only where some other disease of the respiratory organs, or some heart affection, perpetuates the irritation of the mucous membrane. Here the two diseases, thus coexisting, go on reciprocally aggravating each other. In advanced life, after the grand climacteric, chronic catarrh is one of the commonest ailments, being wont, with few interruptions, though frequent fluctuations in degree and extent, to abide until death. It is remarkable that senile catarrh, by determining a continued irritation to the respiratory organs, causes morbid predisposition to unfold itself there,—or disease, elsewhere existing, to throw itself upon the lungs. Thus in the very individuals, who in their youth had been scrofulous, but then escaped,—perhaps even recovered from tubercular disease *by means of* repeated catarrh and its sequelæ, bronchial dilatation and pulmonary emphysema,—we find, in the decline of life, catarrh productive of tubercular development, and even of phthisis. We have already seen what an ascendancy gout exercises over catarrh. In like manner hemorrhoidal disease,—which at the age of manhood is prone to manifest its periodical exacerbations by hemorrhage from the rectum,—sometimes gives rise, under the influence of catarrhal irritation, to repeated bronchial hemorrhage. In females, at the period of menstrual decline, catarrh probably leads to similar accidents.

In advanced age, and where the respiratory organs have suffered, in a high degree, the changes to be hereafter described, chronic catarrh sometimes proves directly fatal, through profuse mucous secretion, and material impediment to the oxygenation of the blood, under the accompaniments of general emaciation, prostration of strength, hectic fever, and frequently of general dropsy.

# CHAPTER V.

## INFLAMMATORY DISEASE OF THE AIR-PASSAGES.

### SECT. I.—INFLAMMATION OF THE EPIGLOTTIS (ANGINA EPIGLOTTIDEA.) ŒDEMA OF THE GLOTTIS (ANGINA ŒDEMATOSA.)

THE two names above specified have by Albers (Pathologie und Therapie der Kehlkopfskrankheiten und Erläuterungen zu seinem Atlas) and others, been applied to two distinct species. It would, however, appear more eligible to consider them under one head, since they generally occur together, and arise from the same anatomical peculiarity, namely, the presence of a considerable layer of loose cellular texture beneath the mucous membrane,—a peculiarity not met with, in the air-passages, beyond the glottis. Both varieties, moreover, present nearly the same train of symptoms, and seem dependent upon the same pathological conditions.

The inflammation assails the mucous membrane above the glottis, and particularly those of its folds which unite the epiglottis, on the one hand with the root of the tongue and the arches of the palate,—on the other, with the larynx, especially laterally, in the direction of the arytenoid cartilages,—these folds being, as is well known, very lax and moveable, and susceptible of great extension.

The disease may seize at once, in consequence of violent catarrhal irritation, upon the above spot,[1] and either confine itself to that, or encroach upon the vicinity;[2] or it may result from the extension of an acute or chronic inflammation, already affecting neighbouring parts.[1] Thus a suppurating tonsil may give rise to this fatal affection; or erysipelas of the external skin, (Bouillaud's second case,) or, again, traumatic inflammation consequent upon surgical operations performed above the larynx.[2] In other instances the main inflammation is rather of a chronic character, or at least latent and insidious in its course, so as scarcely to be cognizable during life; thus the results of previous dissections are all that the practitioner has to guide him, in forming his diagnosis of this dangerous malady. To this category belongs the œdema of the glottis, in which tubercular degeneration and other forms of laryngeal phthisis occasionally terminate, as also that consequent upon so-termed parotid tumour, and upon laryngeal suppuration, during the progress of typhus fever. In a case which came under my notice, of cancerous affection, with caries of the inferior maxillary and hyoid bones, inflammation with œdematous infiltration of the epiglottis, was thus developed. To some such seisure, the cases of sudden death occurring during some slow process of degeneration, are probably often referrible. But the disease in question may follow in the same latent manner in the train of other organic diseases, remote from its own seat, as shown by a case of Louis, (Mém. Anatomico-Pathologiques, p. 359,) where, in a cachectic youth, an hepatic abscess was followed by fatal suppuration about the fauces, with sero-purulent tumefaction around the glottis. Finally, œdematous infiltration may be caused by mere mechanical hinderance, as tumours, which, by pressing upon blood-vessels, prevent the return of blood. It was probably from his having observed cases of this latent kind, where the disease resulted rather from exhaustion,—diminished vascular and nervous energy,—than from inflammatory action, that Bayle was induced to characterize œdema of the glottis as essentially uninflammatory.—The disease is rare, and appears to occur only in the adult, but not in one sex more than in the other. Inflammation of the epiglottis sometimes betrays itself during life, under the aspect of a conical, dark red tumour, (not unlike the glans penis,) projecting behind the root of the tongue. The following are the appearances after death : The whole of the mucous membrane between the root of the tongue and the glottis, is uniformly tumefied, so that the outlines of the epiglottis and arytenoid cartilages, together with the numerous folds and recesses in the vicinity of those parts, have become effaced.

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[1] Home (Med. Chir. Trans. vol. iii.) Mainwaring (Med. Facts and Experim. 1791.) Marsh (Dublin Journ. of Med. Science, vol. xiii.)

[2] Morgagni (De sed. et caus. morb. Epist. iv, 26; xxii, 24, 25; xliv, 13.) Bayle (Mém. sur l'angine œdémat. 1815.) R. Froriep. (Klin. Kupfertafeln. pl. 64.) Cruveilhier (Anat. path. livr. v, pl. 2.) Legroux (Journ. des Connaiss. Méd. Chir. Sept. 1839.)

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[1] Morgagni (l. c. Ep. iv, 24.) Bouillaud (Arch. Gén. de Méd. vol. vii, p. 174; first and third case,) &c.

[2] See Stokes (on Diseases of the Chest.)

This is the result of inflammatory effusion into the interspaces of the loose cellular texture, subjacent to the mucous membrane. In proportion to the intensity and duration of the inflammatory process, this exudation is sometimes of a purely serous and liquid nature, so as to flow away upon incision; sometimes blended with coagulable materials, and jelly-like; sometimes again mingled in various proportions with pus; sometimes wholly purulent. Hence the tumour, which is always soft, lax, and tremulous, like jelly, varies greatly in colour, being of a pale or of a reddish yellow,—sometimes of a dingy yellow or grayish white, and more or less opaque,—but for the most part superficially dotted with red. The swelling being dependent upon infiltration of the submucous cellular texture, cannot extend to the inferior surface of the epiglottis, because, there, no layer of cellular tissue exists; hence the epiglottis has the aspect of having both its lateral edges bent over towards its nether surface, so as to form a narrow perpendicular groove, which is sometimes almost covered by the overhanging tumour. Neither does the swelling extend to the vocal ligaments ( so that the term *œdema glottidis* is not strictly correct) ; but the tumour, hanging down on each side, and in size often exceeding a pigeon's egg, overlays the glottis in such wise as to leave but a narrow opening towards the posterior part which allows the column of air to pass out during expiration, but is closed up by any attempt at inspiration. In no example recorded did the œdematous infiltration beneath the mucous membrane show itself in any marked degree beyond the glottis. The inner surface of the larynx was reddened, puffy, and covered with a puriform mucous layer. Galen's (Morgagni's) ventricles were always more or less implicated in the submucous tumefaction. The mucous membrane of the affected parts is always variously changed. Its surface is rough, its epithelium partly thrown off in single scales and tufts, or raised by serous fluid;

it exhibits here and there blood-specks, or scattered bright-red vascular streaks; it is moreover easily separable, and, when separated, friable. The neighbouring muscles, especially the arytenoid, are sometimes unaltered, at other times saturated in like manner with the effused fluid, in which case they have become blanched, or undergone yellowish or grayish softening. Where the disease is of catarrhal origin, the fauces and the palatine region are generally found simultaneously inflamed, and the tonsils destroyed by suppuration. The inflammation commonly ends where the pharynx passes into the œsophagus, and again at the glottis; yet there have been instances (see Bouillaud's first case) of its pervading the whole of the air-passages and attacking portions of the lungs.

Œdema of the glottis, when at all violent, commonly proves fatal. Inflammation of the epiglottis *alone* is perhaps more susceptible of cure, in which case the organ appears to undergo hardening, and to shrivel, and diminish in size.[1]

## SECT. II.—INFLAMMATION OF THE LARYNGEAL CARTILAGES. PERICHONDRITIS LARYNGEA.

A violent form of inflammation which assails the larynx in its totality and is not confined to, perhaps only slightly affects the mucous membrane, appears to have its seat more especially in the perichondrium of the laryngeal cartilages. But the inflammation not having here, as in the foregoing disease, a considerable layer of cellular texture for the reception of its products, ere long induces suppuration and extensive destruction of, with separation of the mucous membrane from, the cartilaginous framework. In this manner frequently arise collections of pus, known under the denomination of laryngeal abscess, and which in the sequel lead to regular laryngeal phthisis, quite independent of constitutional tubercle.[2] In the majority of cases the perichondrium of a single cartilage,—for the most part the cricoid,—is principally affected, the rest suffering only in a secondary way; sometimes the destructive process takes an inward track, throwing itself upon the mucous membrane; it may, however, also proceed outwardly, and spread, between the laryngeal muscles, to the external skin, or affect the cellular tissue intervening between the air-passages and the pharynx, opening a passage for the pus in either direction. In this manner abscesses may form either beneath the external skin of the throat, or else behind the larynx; these, after a train of suffocative symptoms, more or less imminent, lead to perforation of the œsophagus, the trachea, and the larynx, proving either directly fatal or productive of momentary relief.

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[1] See Home, (l.c.), and Albers (Erlaüter. zu dessen Atlas der Path. Anat. part ii, p. 7.)

[2] Cases in point have been collected by Albers in his treatise on the Diseases of the Laryngeal Cartilages (see Gräfe und Walther's Journal, vol. xxix, fascic. i, p. 6–14.) To these are to be added two cases by Ch. Bell (Surg. Observat. London, 1816); several by Sachse (Ueber Kehlkopfs und Luftroehrenschwindsucht, p. 133); and one by Mohr (Beyträge zur Pathol. Anat. 1838, p. 28.)

With this evacuation of the abscess, however, the disease is by no means at an end; a most important feature being here about to ensue, namely the consecutive affections of the cartilages. For these becoming, by dint of suppuration, stripped as through maceration, of the submucous cellular texture and of their perichondrium, lose all organic connexion, mortify, and go on irritating as foreign bodies, until they have become ejected and removed, either piecemeal or entire. Ere this happens, however, death by suffocation commonly takes place, either from the larynx, thus robbed of its due support, falling in, or else from fragments of cartilage projecting into the air-passage and closing the aperture of the glottis, or, in fine, from the abscess continuing, amid profuse suppuration, to enlarge to such an extent as ultimately to intercept the passage of air into the lungs. Occasionally a rapidly fatal attack of œdema of the glottis will supervene.

In these cases we find a purulent cyst, of greater or less volume, projecting, either somewhat into the larynx, or else outwardly; the surrounding cellular texture is of brawny hardness, forming a perfect bond of union to the various parts, between which isolated fistulous channels are sometimes found passing. The muscles are drenched with serum, and blanched, or softened, and slate coloured. Blood-vessels and nerves are not clearly distinguishable (Bouillaud. Journ. complém. du Dict. des Sc. Méd. vol. xix, p. 3). The abscess is lined with a soft, velvety, reddish or grayish membrane, and filled with pus,—becoming fetid when exposed to the air,—otherwise well conditioned. The affected cartilage appears on all sides separated from its investment, attenuated, or riddled, or fallen to pieces; it looks as if eroded, is rough, softened, and sometimes discoloured. Porter (The Surgical Pathology of the Larynx and Trachea) found, in two instances, the portions of cartilage which had come in contact with air, partly in a state of blackish softening, like decayed leather, partly of a brown colour, and shrivelled up into a horn-like substance. Where the disease is acute, not consequent upon a chronic affection of the larynx, and not occurring in very aged subjects, the necrosis is never preceded by ossification of the cartilages. The reverse holds in tubercular phthisis of the larynx.

*Recovery* now and then takes place, even after the elimination of necrosed fragments of cartilage. This probably happens only where the abscess has been able to discharge freely through large openings, and where the perichondrium has not entirely sloughed away. To judge from analogy, the fragments of cartilage ejected would be replaced, though imperfectly, by a bony deposit. Examples of this kind, referrible to genuine perichondritis, are not indeed on record,[1] but Gendrin[2] has related one instance, and Trousseau and Belloc[3] another, in which tubercular necrosis had been compensated for by ossification in and beneath the perichondrium. Actual regeneration of cartilage does not appear ever to take place.

The disease under consideration occurs, in rare instances, idiopathically, as a catarrho-rheumatic affection; nor is it upon the whole frequent. The majority of known cases are secondary, having been either the sequel or the attendant of smallpox, typhus fever, (Bouillaud, Cruveilhier,) and constitutional syphilis. I have myself seen it but once as an idiopathic affection, though repeatedly as the result of typhus fever. Out of thirteen examples of perichondritis, collected by Albers (l. c. p. 14), eleven were in men and two in women. The individuals were respectively between the 9th and the 64th, but most frequently between the 25th and 40th years.

It may be stated in conclusion, that inflammation of the parts subjacent to the mucous membrane of the windpipe, in rare instances terminates, not in suppuration, but in thickening and fibro-lardaceous degeneration, productive, it may be, of stricture of the larynx, and eventual suffocation.

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[1] Unless we might reckon as such the case of White (American Med. Libr. &c. vol ii, p. 33), where the cartilages were partially ossified, and a new bony plate had formed upon the thyroid cartilage.

[2] Anatomical description of Inflammation.

[3] Mém. de l'Acad. Roy. de Méd. vol. vi, p. 142.

Albers (l. c. pp. 4-6) has recently demonstrated that cartilage is itself susceptible of idiopathic inflammation and suppuration. In the acute form, this takes place only where partial or total ossification has preceded; whereas chronic inflammation may attack unossified cartilage. The cricoid cartilage appears almost exclusively prone to the disease; in one instance it contained, within its substance, an abscess which at its upper margin had opened into the pharyx. The perichondrium, with the cellular texture, appear to become subsequently implicated, so that two abscesses form, one within, the other without the cartilage, and afterwards tend to unite. In its symptoms and pathological characters no difference is observable between this disease and perichondritis.

## CHAPTER VI.

### EXUDATIVE INFLAMMATION OF THE AIR-PASSAGES.

### EXUDATIVE LARYNGITIS,—TRACHEITIS (CROUP),—BRONCHITIS.

1. *Exudative laryngitis.* Mucous membranes are naturally but little susceptible of this kind of inflammation. It is therefore, upon the whole, rare, except in the air-passages, where it constitutes a separate species. Its distinctive characters, according as it affects the larynx, the trachea, and the bronchia, are immaterial, in so far as morbid anatomy is concerned, though all important as regards the symptoms and general pathological import. Exudatory inflammation appears, in the respiratory passages, to spread, invariably, from above downwards, never in the opposite direction; so that, when commencing in the bronchia, it can only descend to the pulmonary cells, never mount to the larynx. This is the more worthy of note, inasmuch as there appears to be a tendency in the disease to descend,

in a like manner, with advancing life. Thus in children, it is in the larynx and trachea that it is most strongly marked, whilst in adults its occurrence elsewhere than in the lesser ramifications of the bronchial twigs, is but an exception.[1] When the mucous membrane, in consequence of inflammatory action, throws out coagulable matter on its surface, it presents the same characters as the serous membrane, the chief difference being, that the shut sacs of the latter hermetically close the inflammatory product, and must further elaborate it, so that it may be either converted into a homogeneous substance, or removed by absorption, whereas the products of the mucous membrane come directly into contact with the external air, and find a ready vent. In drawing this comparison, the question may be propounded, whether the lining of the more delicate bronchial twigs, and of the pulmonary cells, which in the *absolute* majority of cases are the seat of plastic effusion, may not bear some analogy, in point of structure, to serous membrane. At any rate, the muciparous glands are not found in the bronchia beyond a certain depth; nor can the presence of an epithelium, or even of ciliary organs, constitute any decisive ground of distinction from serous membranes, now that we are acquainted with Henle's researches on this subject.

2. *Tracheitis, Croup* (*angina membranacea*), is in most instances developed from a preexisting catarrhal state, generally of brief duration. No sooner does the catarrhal irritation merge into inflammation, than the plastic lymph is thrown out, and the parts immediately suffer that disturbance of nervous energy which results from all violent inflammations. In croup this species of disturbance is particularly manifested in the impaired action of the laryngeal muscles, more especially of those which serve to open the glottis.[2] According to the best authorities

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[1] The above assertion would acquire further strength from a confirmation by other pathologists of Billard's remark (Arch. Gén. vol. xii, p. 555), that, in infants at the breast, croup is exceedingly rare, but that false membranes are frequent, at that age, in the nostrils, during coryza, although they never extend to the larynx.

[2] This disturbance of muscular activity is quite analogous to (though far more perilous than) impairment or stoppage of the respiratory movements, in one-sided pleurisy, &c. In croup, emetics would appear to answer a twofold indication: first, that of removing the mechanical impediment; and, secondly, that of creating a new action in the nerves of the affected part.

(Stokes and J. A. Albers) death is, for the most part, rather due to this inflammatory spasm, than to mechanical hinderance; and it is probable that those extraordinary remissions, which the last-mentioned writer [1] has stated to have been *erroneously* regarded as intermissions, may in a great measure depend on the same thing.

The plastic exudation forms with great rapidity. It has various gradations; it consists sometimes of a tenacious mucus, wherein are suspended thin membranaceous flocculi; sometimes of an extraordinary quantity of a yellowish or reddish, ropy, and here and there turbid, fluid; sometimes, again, of a layer, resembling, both in colour and consistency, that which settles upon scalded cream; and sometimes of a firm and tough false membrane of considerable thickness. There are examples of this false membrane extending in a nearly uniform character throughout the greater portion of the air-passages, pervading the arborescent ramifications of the bronchia, and even descending from the fauces into the œsophagus. Such instances are, however, rare; the plastic mass being found, for the most part, no farther than the trachea and its principal branches, where it adheres firmly in the shape of a connected membranous tube; lower down it diminishes in consistency, loses the tubular form, and assumes that of a viscid, yellowish-white layer of mucus, or else of a solid plug, made up of a jelly-like, tenacious, and yellowish, or a dull-white, fissured mass, resembling boiled white of egg. In many cases the bronchial twigs are replete with common mucus, or catarrhal secretion, which being mechanically kept back by the plastic impediment above it, thus tends to promote suffocation. In the larynx, are mostly found individual, irregular and loosely adherent fragments; these are likewise frequent beyond the glottis and in the throat, more particularly about the tonsils. The depth of the adventitious membrane is extremely variable, but nowhere exceeds three lines, being commonly thickest upon the posterior wall of the trachea; its density is not proportionate to its thickness. Its hue is more or less of a grayish white, or of a dingy dull yellow. The exudation consists mainly of fibrin blended with mucus in various proportions. By incineration Schwilgué

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[1] J. A. Albers (Commentatio de tracheitide infantum, &c. Lips. 1816. pp. 19, 25, 33; aliisque locis.)

(Du Croup aigu des Enfans, Paris, 1802,) detected in it a small quantity of carbonate of soda and phosphate of lime. Laennec is wrong in stating it to be formed by the consolidation of purulent fluid; because, generally speaking, the plastic exudation contains but a very small number of pus-globules.

At the commencement, the membranaceous product adheres with tolerable firmness to the mucous membrane, and displays a nearly uniform character; but in proportion as the inflammation abates, a grayish and sometimes tenacious, at other times mere watery mucus becomes deposited beneath it, the adventitious layer loosens, and is expelled, amid violent fits of cough, either in fragments, or else in perfect cylindrical and arborescent masses, representing the parts where it has formed. Where, however, the false membrane cannot be entirely expelled, the whole mass sometimes liquefies into a grayish or puriform mucus; or becomes attenuated, and perforated with little holes, without separating; so as occasionally to remain long fastened, like a network, to the surface of the mucous membrane. In such cases suffocation may ensue some days after apparent, amelioration, owing to remnants of the false membrane becoming detached, and clogging up the aperture of the glottis.

Croup usually finishes its course with a single process of exudation and its sequelæ; there are, however, examples of the inflammation exhausting itself only after the formation of a second, or even a third adventitious membrane. After one attack, there generally remains, for a length of time, a proneness to plastic exudation. It is stated by J. A. Albers, that Olbers met with croup recurring nine times in the same child.

With respect to the susceptibility of the adventitious membrane to enter into organic union with its matrix, various opinions formerly prevailed. J. A. Albers (l. c. pp. 28, 108) believes, that in many cases a cure is effected through adhesion of the false, with the mucous membrane; yet he denies ever having perceived blood-vessels in the former, and maintains that others have deceived themselves in this particular. He grounds the above opinion solely upon one of the preparations in Sömmering's museum; but as this would appear to be the only authentic example of the kind upon record, and as nothing analogous has been met with since, it ought to be regarded as but a striking exception; even Guersent (Nouveau Dict. de Méd. art. Croup) speaks of organization, only as a *possible* thing. Carswell (Fasc. Inflammation)—and with him probably most modern writers—utterly denies the possibility of mucous membranes generating organizable false membranes;—he could never detect a vestige of vascular formation or of true adhesion therein.[1] The *effort* at assimilation is, however, in some instances very perceptible in the appearance of stellated ecchymoses and bloody streaks on the surface of the false membrane facing the mucous membrane. The filamentous bands sometimes found between the plastic exudation and the mucous membrane, consist merely of delicate fibrous threads, which dip into orifices of the muciparous glands.

The mucous membrane subjacent to the effused mass, displays certain deviations in character. It is for the most part reddened, sometimes of a crimson, sometimes of a vermilion hue, or, again, violet coloured, or even blackish, in specks or spots, which latter coalesce here and there into irregular longitudinal stripes. In other cases it is of a paler and more equable rosy tint. At a subsequent period, the inflammatory redness gradually disappears, and the mucous membrane has regained its natural pale colour, which circumstance has led to a disbelief in the inflammatory nature of croup. This notion is, however, confuted both by anatomico-pathological facts, and by the experiments of J. A. Albers; who, by exciting artificial inflammation, produced spurious membranes within the air-tubes of animals. In certain forms of exudation, consequent upon inflammation of the serous membranes, the latter in like manner present no appearance of redness: this recurs only at a later period, in the shape of lively vascular injection, preparatory to organization. The mucous membrane is generally brittle, friable, and thickened; in rare instances, in a state of gelatinous softening. Whatever may be its colour, its surface is invariably more or less rough, like the under side of woollen cloth. The subjacent cellular texture is infiltrated with serous, gelatinous, or opaque fluid; Guersent even detected in it little collections of pus.

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[1] Hence there appears little ground for Villermé's assumption, that the peculiarity of voice, which long remains after recovery from an attack of croup, is caused by organized remnants of the adventitious membrane; it is probably rather referrible to protracted impairment of innervation, and to the altered texture and function of the mucous membrane.

Similar adventitious membranes have been found in the œsophagus, in the fauces, in the nostrils (even in the frontal sinuses), and in the mouth; sometimes as though the disease had commenced at the palate, and descended from thence into the air-passages. The lungs generally do not collapse, are dark-coloured, turgid with blood, and occasionally exhibit isolated patches of inflammation. The lymphatic glands about the neck and bronchia are often enlarged, grayish-coloured, and softened, or else dark red, and indurated. Both sides of the heart contain a quantity of dark coagulated blood, together with fibrinous concrements: as in most instances of death from suffocation, the brain shows a congested state of its blood-vessels, and serous effusion upon its surface, and within its ventricles.

Croup is a disease of childhood, and occurs principally between the second and the twelfth year, before and after which it is rare.[1] According to most pathologists (J. A. Albers, for example) it is more common in boys than in girls. Jurine states that, during a period of eighteen years, thirty-seven girls and fifty-four boys died of croup at Geneva. In southern latitudes it is far less frequent than in northern. J. A. Albers says that from forty to fifty cases are known to occur annually at Bremen, whilst, in the south of France, the proportion is far smaller, and less still at Leghorn.

Nearly akin to croup is the membranaceous inflammation consequent upon violent implication of the air-passages, during *measles*. It is to be regarded as a consequence, less of any particular malignancy in the prevailing epidemic, than of the catarrhal tendency of the exanthema.[2] *In scarlatina,* on the contrary, it is almost exclusively in the most malignant form of the exanthema, where the whole mass of the fluids has become vitiated, that membranaceous inflammation of the cavity of the fauces, extending, it may be, to beyond the larynx, supervenes. The exudation differs from that of croup in being less consistent and less uniformly spread over the involved part, taking rather the form of a friable, superficial granular growth, or else of a puriform, soft, grayish false membrane ; it is usually poor in fibrin, and prone to decomposition. The subjacent mucous membrane is likewise discoloured, and of pultaceous softness.—Croup-like symptoms, when manifested during *smallpox*, are not due to plastic exudation, but to vesicular or pustular elevation of the epithelium, just as diphtheritis or aphthous disease, when thrown upon the air passages, consists in partial shedding of the epithelium by means of a serous, or a whitish, coagulable secretion. A rapid and diffuse incrustation of the epithelium, with consecutive exudation of coagulable substances, is produced by the action of *caustic acids* in a fluid or gaseous form. The effect would appear to be particularly marked in the instance of sulphurous acid. J. A. Albers (p. 68) narrates two cases of suffocation with croup-like symptoms and membranaceous expectoration, brought on by the inhalation of chlorine vapour.

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[1] Of the cases adduced by Louis (vide infra,) of croup in the adult, two,—the first and seventh,—are alone admissible here. Nor are all the examples cited by J. A. Albers veritable croup.

[2] The most strongly marked case I ever met with of tubular expectoration from the trachea and bronchi, occurred during the prevalence of a very *mild* epidemic of measles.

Exudative inflammation of the upper portion of the air-passages, is very seldom idiopathic in the adult. (See Louis, Du Croup chez l'adulte, in the Mém. Anatomico-pathologiques, pp. 203-252.) In the majority of the few authenticated cases, the individuals were, when thus assailed, already labouring under some other disease, either acute or chronic. They were, generally speaking, not persons of a robust habit, but such as had been previously reduced by misery, or afflicted with typhus, pulmonary phthisis, or chronic pleurisy, and the like. Here the exudation upon the respiratory mucous membrane was either a complication, arising out of incidental causes, or only a symptom of a general decomposition of the fluids, with complete alienation of the formative process and of nervous energy. The false membrane always commenced at the palate and fauces, now and then in the Schneiderian membrane, and, gradually decreasing in consistency, proceeded from thence to invest, on the one hand, in an unlimited manner, the interior of the air-passages,—on the other, the pharynx, the œsophagus, and, in one instance, even a portion of the stomach. The crowing tone of the voice and of the cough usual in children was invariably wanting,—a circumstance due no doubt to the greater width of the larynx and of the glottis in the adult.[1] Death seemed to be rather the consequence of nervous palsy than of mechanical obstruction. It would seem, therefore, as above stated, that croup in the adult is scarcely to be regarded as constituting an individual species, but for the most part as a merely accidental disease, resulting from a concurrence of pathological relations.

3. *Exudative bronchitis* is, on the other hand, not unfrequent in the adult. Its extent is, however, limited; for the genuine croup-like and rapidly destructive kind, which, commencing at the partition of the trachea, and spreading on one or both sides throughout the majority of the bronchial branches, without necessarily reaching the pulmonary cells, is almost exclusively confined to children, and in them occurs rarely without implication of the trachea; in the adult it is met with only as a rare exception. In the latter, as will be shown hereafter, it is almost always contemporaneous with catarrhal pneumonia, and in that case alone pervades the whole system of the bronchial tubes pertaining to a pulmonary lobe. In ordinary bronchitis, or inflammatory catarrh, even when diffused through the greater portion of a lung, a few scattered branches of small diameter are alone found clogged with white plastic coagula, down to the still more diminutive twigs, whilst the remainder contain merely the usual, fluid, catarrhal secretion. All persons, children as well as adults, once afflicted with exudative inflammation of the bronchia, are extremely prone to relapses, just as in croup.

With reference to the anatomy of plastic bronchitis, there is nothing to add beyond what has been stated as to the behaviour of the bronchial tubes in croup. Of late, Reynaud,[2] and more particularly Stokes (l. c.), impressed with the notion that the lining membrane of the minuter branches presents nearly the same anatomical characters, and consequently the same pathological relations, as the serous membranes, have included, under the head of bronchitis, obliteration of the bronchial tubes. Plastic exudation within the bronchia should accordingly lead to cohesion of their inner surface, and their total obliteration, followed by atrophy and shrivelling of the texture of the lung. This, however, requires confirmation. In the first place, it is necessary to distinguish between simple obstruction, and complete obliteration. The former no doubt does occur, and where the obstructing cylinder is firmly impacted, abides the longer, owing to the want of a forcible backward current of air acting upon it. This is especially evident in those cases of hooping-cough, which terminate fatally through plastic bronchitis supervening at one or at several points. Here the lung presents various conditions: individual portions are distended by recent emphysema, and remarkably pale and exsanguineous;—other, and smaller portions appear on the other hand as if sunken,—and are deep, almost violet-coloured,—not at all crepitant, and saturated with dark blood;—they are tough and flabby, and sink in water. The bronchial tubes, for the most part, evidence catarrhal irritation, and the lesser twigs are clogged, in the last-mentioned localities only, with a solid white plug, so as to intercept the transit of air to the pulmonary cells, and render the latter vacuous and collapsed. The same condition is sometimes, though more rarely, observed in primary plastic bronchitis. Examples, in which polypoid growths, after long resistance, are eventually expelled by a violent fit of cough, constitute chronic plastic bronchitis, mentioned by some of the older writers,—Cheyne amongst the rest.[1] A perfect, cord-like obliteration, or thorough organic blending of the effused substance with the bronchial parietes, I have never been able to discover. Wherever there was complete and decided obliteration, there was far more reason to apprehend that it proceeded from the parenchyma of the lung, and that it was caused, for the most part, by tubercular disease, especially during the process of cicatrization, though now and then by partial, inflammatory disorganization of the lung itself.

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[1] In one instance, related by Lynch and Dawson (Dublin Journ. Sept. 1838), the sound emitted in coughing resembled the bark of a little dog; both the trachea and the glottis were, however, found unusually narrow.

[2] Mém. de l'Académie Roy. de Méd. vol. iv, 1835, p. 117.

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[1] North (Lond. Med. Gaz, May 1838.) Cane (Dubl. Journ. May 1840.) Brummer (Casper's Wochenschrift, 1841. No. vi.)

# CHAPTER VII.

## CATARRHAL PNEUMONIA,
## (BRONCHOPNEUMONIA, PNEUMONIA NOTHA.[1])

In many instances this variety differs from ordinary pneumonia in nothing else than in the peculiar mode in which it is developed and diffused. In other cases, on the contrary, there is a wide difference in the condition of the pulmonary texture, in the quality of the inflammatory product, and in the phases which the latter undergoes. When near the surface of the lung, the diseased portions rather sink in than bulge out, and are, if recent, somewhat incompressible, but of progressively increasing flabbiness where the disease has been protracted. They are, at the outset, of a dingy, blue reddish hue, which, however, becomes paler by degrees, changing first to a brown-red, and eventually to a yellow-brown. The texture thus altered is altogether impermeable, and soft, though less friable than in hepatization. At the beginning, it is saturated with a turbid reddish fluid, which contains numerous blood-globules; by-and-bye it becomes more and more opaque, gradually passing into grayish,—as though it were mixed with purulent mucus, and ultimately assuming altogether the aspect of a pale, yellowish-brown, puriform fluid. This, when examined by the microscope, is seldom found to be amorpho-granular; it usually contains granule-cells in predominant numbers, but often intermingled with exudation-cells, and sometimes with a proportion of pus-globules. When a large portion of a pulmonary lobe becomes thus disorganized, which is, however, rare, it is found shrivelled, lax, moist, of a yellow-brown hue, and wholly devoid of air,—resembling a wet rag. This kind of catarrhal pneumonia appears peculiarly calculated to produce obliteration of the pulmonary texture, with permanent exclusion of air therefrom, and consequent general dilatation of the several branchlets of the bronchia engaged.

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[1] What Reil describes under this name is simple bronchitis.

Catarrhal pneumonia invariably originates in catarrhal affection of the air-passages, which, having, in more or fewer of the different bronchial ramifications, attained an inflammatory height, ultimately proceeds, in this wise, to implicate the pulmonary cells. This form of pneumonia frequently puts on the vesicular or lobular type, and involves an *entire* lobe only when every canal of the latter disseminates the inflammation in the manner described, or when the vesicles of one lobule infect those of another, contiguous lobule. Frequently individual patches, in the midst of a lobe, are found consolidated, in consequence of catarrhal inflammation (central pneumonia.)

The disease—and more especially the peculiar form just alluded to—is, in its different phases, a tolerably frequent result of pertussis or severe catarrh, and is scarcely ever missing in fatal cases of bronchitis. In like manner, inflammation of the parenchyma of the lung often displays the peculiarities described, when occurring during the progress of smallpox or measles.

It will be seen from the above, that catarrhal inflammation constitutes one of the most insidious forms of pneumonia. It is extremely difficult—sometimes, indeed, impossible—to make out the physical signs; partly, because the patches are of small extent, situate in the midst of a lobe, and surrounded with healthy parenchyma; partly, because the physical signs of inflammation are, in a great measure, masked by the diffuse catarrhal affection. It is proved by cadaveric inspection, that in many cases of catarrh, supposed to have put on a typhoid character, a partial pneumonia had reached its second stage, without having been suspected during life. Certain catarrhal epidemics are peculiarly apt to superinduce pneumonia, as shown in various places in the instance of influenza.[1]

A further distinction between catarrhal and ordinary pneumonia, consists in the presence of plastic exudation within the bronchia. Thus, on narrowly scrutinising the hepatized lobules, which now are distinctly circumscribed, now gradually pass into a parenchyma drenched with bloody serum, we find all the bronchial tubes, immediately connected with that part, filled with a white and solid, or else yellowish and softish, true fibrinous coagulum, which, in the larger branches, passes at length into ordinary bronchial mucus; but, in the opposite direction, may often be traced down to the effusion which fills the pulmonary cells. These plugs are for the most part firmly adherent, being loosened only in the wider tubes, where a layer of thin mucus separates them from the internal membrane.[1]

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[1] Out of 183 cases of influenza which occurred at Paris in 1837, pneumonia was present in 40. See Landaus (Arch. Gén. Avril 1837.)

It would appear desirable here to revert once more to the question before touched upon (see pneumonia), concerning the real seat of inflammation in the texture of the lung; a question which has given rise to much discussion,[2] and still seems open to farther investigation. The parenchyma of the lungs being considered as composed of blood-vessels, of bronchial-tubes together with their terminations, and of cellular texture,—the network of capillary vessels,—the pulmonary cells,—the cellular tissue—are each in turn looked upon as the true focus of inflammation. It would, however, be difficult, in support of any one of these views, to demonstrate, anatomically, either an actual limitation to those textures, or the line of demarcation between pneumonia on the one hand, and bronchitis, pleurisy, and phlebitis of the lung on the other. For if the interstitial cellular tissue be held to be the real seat of pneumonia, the latter must needs be restricted to the parts immediately subjacent to the pleura, to the indentations that mark the confines of the pulmonary lobes, to the course of the larger bronchial and vascular trunks, and, finally, here and there, to the interlobular spaces,—in which situations alone a small proportion of cellular tissue is really cognizable. Elsewhere, it has ceased to be cellular tissue, and constitutes, in effect, bronchial, cellular, and vascular parietes. Nor can the capillaries be held to be the exclusive seat of pneumonia, at least, not apart from the pulmonary cells, which (according to E. H. Weber) have a capillary network so dense, that the diameter of the vessels exceeds the intervals between them. For similar reasons,—irrespectively of the stronger arguments derived from the microscopic characters of inflammation,—the *solitary* affection of the coats of the cells, without implication of the capillaries, must be deemed impossible. Nor could the notion be consistently entertained that any marked difference between inflammation of the bronchia, and of their ultimate terminations (the pulmonary cells), indicated precisely where the one ended and the other began. Those questions and doubts have obviously arisen from a false interpretation of some of the brilliant results of recent microscopic research, and forcibly point to the neglected condition of general pathology.

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[1] This compartment of the bronchia, in certain cases of pneumonia, was first pointed out by Lobstein, and subsequently described in detail by Reynaud (Mém. de l'Acad. Roy. de Méd. vol. iv, p. 155), and by Stokes (l. c.)

[2] See, for example, Cruse (über die Bronchitis der Kinder—on the bronchitis of children. 1839.)

Every inflammation, whether of dynamical, of mechanical, or of chemical origin, is mainly conditional upon a peculiar alteration in the contents of the capillaries, and in the parenchymatous fluid; the elementary structures are changed secondarily. (Compare Gluge, Anatomisch-microscopische Untersuchungen, &c. 1839, p. 33.) The product of inflammation is thrown out into every interspace at hand, and influences the involved textures differently, passes through different phases of transformation, and terminates in different ways, according as the form and degree of the inflammation vary. In the lung all inflammation, at whatever point and from whatever cause developed, must, therefore, needs originate in the capillary system, implicate every individual texture, and fill each interspace with its products. We have seen, however, that the parenchyma of the lung may become primarily inflamed. Here the capillary system of the *functional* vessels is alone actively engaged, the true functional portion of the different parts which constitute the pulmonary mass, being alone primarily affected with the inflammation, whilst whatever lies beyond the immediate range of the capillaries of the pulmonary artery (be it bronchial tubes, blood-vessels, cellular tissue, or patches of pleura,) is only accidentally implicated. This, however, occurs the more readily from there being no absolute circumscription of the above capillary network, which, on the contrary, inosculates, at many points, with the vessels of nutrition, so as very frequently to involve the lesser bronchia in the inflammatory process. In other instances the texture of the lung becomes inflamed, not originally, not in its totality, nor in its expansion as a respiratory organ, but through its contiguity with other parts. Where this proceeds from the pleura, the principal seat of the inflammation is generally the cellular tissue of the pulmonary substance, as being the texture nearest allied to serous membrane. Here the part chiefly affected is the periphery of the lung, down to the indentations of the lobes, and along the ramifications of the vessels and of the bronchia;—the pleura readily peels off, and the lobes and lobules stand out, as though prepared by maceration. This is by some termed "cortical pneumonia," by Stokes (l. c.) "dissecting abscess." Where the inflammation arises in the branches of the pulmonary artery, it spreads, only within the range of those branches, to several circumscribed patches of the parenchyma,—a condition already described as lobular abscess. (See Phlebitis.) Lastly, we have seen that catarrhal inflammation of the bronchial branches is capable of advancing to the pulmonary cells, and there engendering, in like manner, a more or less circumscribed, lobular pneumonia. In all of these last-mentioned cases, the pulmonary substance may, for the most part, be seen, at a glance, to be only secondarily diseased, the marks of inflammation being confined to the range of parts greatly disordered. This is, however, less apparent, where such secondary pneumonia (from contiguity or other causes,) has involved an entire lobe; the primary phlebitic or bronchitic affection being here frequently veiled, and no longer to be traced without a very close examination. Where, on the contrary, the proper substance of the lung is the original seat of inflammation, those portions of the pulmonary texture not supplied by the capillary system of the pulmonary artery,—namely, the bronchia, blood-vessels, and pleura, are found implicated only in a subordinate degree.

# CHAPTER VIII.

## ORGANIC SEQUELÆ OF CATARRH.

### SECT. I.—HYPERTROPHY OF THE MUCOUS MEMBRANE, AND OF THE MUCIPAROUS GLANDS.—FORMATION OF DIVERTICULA AND OF POLYPOUS GROWTHS.

1. In all mucous membranes, permanent or repeated sources of irritation are productive of augmented secretion, and eventually of various changes of structure and external form. Such an effect is produced upon the mucous membrane of the air-passages by chronic catarrh. The nature of these changes varies greatly, however, according to the different portion (anatomically speaking) of the membranous structure, and to the different parts of the air-passages involved. Some of these sequelæ are to be regarded as the invariable consequences of catarrh, and are therefore matter of daily observation; others require a peculiar predisposition,—a concurrence of peculiar circumstances,—for their development; are not the immediate consequences of catarrh, or of catarrh alone; and are of rare occurrence.

As we intend devoting separate chapters to the important subject of the dilatation of the air-tubes and air-cells, we shall now proceed to treat of thickening from luxuriant growth of the mucous membrane, the result of increased formative energy, and also of hypertrophy, similarly produced in the muciparous glands, and subject in like manner to peculiar deviations of form.

In chronic catarrh, there is always manifest *thickening* of the *mucous membrane,* without marked alteration in its structure, often, indeed, (in the dead body at least,) without any unusual redness. This thickening frequently exists, in a less degree, within a space of an inch at the extremity of the trachea, and at the commencement of the two main bronchial trunks. Here it produces little inconvenience, until aggravated by a fresh attack of acute catarrh, when the tumefaction may augment so as to obstruct the main tube of an entire lobe, and thus endanger life, (see Andral, Clin. Méd. 4me ed. vol. iii, p. 176.) Such thickening is, however, far more perilous, when at the entrance of the larynx, and when kept up by sustained irritation. It then proves fatal, sooner or later, under repeated suffocative seizures.[1] The surface of the thickened membrane is sometimes smooth, at other times rough, as if stripped of its epithelium. In some instances, however, it appears as if the epithelial layer were itself thickened, the surface becoming finely granular, velvety, or even villous, like the intestinal mucous membrane. (Reynaud.) With the exception of confirmed old age, in which the mucous membrane is found atrophied, and the bronchial tubes dilated, the above preternatural conditions are more strikingly developed in proportion as the individual advances in years.

In rare instances hypertrophy of the mucous membrane may increase inordinately, and ultimately engender *polypous growths.*[2] These polypi, however, are the product, not of simple catarrh alone: they indicate a deeper seated malady. Sometimes they are not restricted to the mucous membrane, but strike root more deeply within the cellular tissue, or in the perichondrium, or other fibrous structures, projecting, after previous ulceration, beyond the mucous membrane. Here catarrh may be the occasional cause or the accompaniment, or be altogether wanting,—the symptoms merely at first simulating those of chronic catarrh. Polypi of the *larynx* are extremely rare; there being scarcely more than 16 cases upon record, 14 of which happened in men. They affect the upper portion of the larynx, and the vicinity of the vocal chords,—these last indeed, and Morgagni's (Galen's) ventricles, appear their exclusive seat. They are either sessile upon a broad base, or are more or less pediculated, being sometimes firm, fibrous, smooth, and roundish; at other times wart-like, clustered, and cauliflower-like; or, again, very soft, vascular, and multilobular. They vary in size, from that of a pea to that of a walnut. The surrounding mucous membrane seems at times perfectly healthy, at others in various degrees inflamed; in a few of the cases it was ulcerated, and the textures, down to the laryngeal cartilages, destroyed. Polypi developed within the ventricles of Galen, occupy their cavity, displace their walls, elevate the vocal chords, and, by ultimately closing the glottis, occasion death by suffocation. In several of these instances the disease ultimately became complicated by œdema of the sub-mucous cellular tissue.

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[1] To this category belong most of the cases described by Trousseau and Belloc (Mém. de l'Acad. Roy. de Méd. vol. vi, 1837, Obs. viii–xv), as simple, non-tubercular, laryngeal phthisis. Some of them were accompanied by chronic inflammation of the mucous membrane with ulceration,—others, by effusion of different kinds into the submucous cellular tissue,—others again, by hypertrophy and purulent softening of the muciparous glands.

[2] The literature of laryngeal tumours, and especially of polypi, is embodied in J. F. H. Albers' works: (Kehlkopfskrankheiten, p. 99; Path. Beobacht, vol. i, p. 101; Erläuterungen z. Atlas d. Path. Anat. part ii, p. 49.)

In the *bronchi* the occurrence of polypi is still rarer. Laennec met with one an inch and a half long, and four or five lines broad, in the left bronchus; it was of a reddish violet hue, of firm fibrous texture, and contained a few small blood-vessels. Older writers have adduced numerous examples of polypi in the air-passages; little faith is, however, due to them, inasmuch as it was customary, in former times, to designate every fibrinous exudation as polypus.

Constitutional *syphilis* is known to predispose to repeated attacks of catarrh, and to consequent thickening and excrescence of the mucous membrane of the larynx. This thickening affects the general texture of the mucous membrane, converting it into a homogeneous, lardaceous substance, which at times materially constricts the larynx. In certain instances (Rokitansky, Oesterr. Jahrb. N.F. vol. viii, p. 441), the disorder extends to the cartilages, producing suppuration, shrivelling, and fatal destruction. The growths are wart-like, sessile upon a broad base, and present, both in form and structure, the greatest resemblance to condylomata. They occur at the epiglottis, at the upper entrance of the larynx, and at the vocal chords—seldom lower down (Otto, Albers); they are mostly small, though capable of attaining a large size, (Morgagni, Epist. xliv, 3,—Rayer, Malad. de la Peau. pl. xv, fig. 21,—Barth, Arch. gén. 3$^{me}$ sér. vol. ii, p. 277.) These condylomata are equally frequent in males and females; they are, however, far less common than genuine syphilitic ulcers of the larynx, which, on cicatrizing, leave the internal membrane, in a like manner, rugged, with irregular folds and protuberances. (Morgagni, Epist. xliv, 15.)

Another consequence of catarrh is *hypertrophy* of the *muciparous glands,* which is met with in the most various grades. Its most frequent form is that of granular unevenness of the mucous membrane on both sides, and on the under surface of the epiglottis, advancing from thence to the vocal chords; or that of puffy tumefaction and irregular filling up of Galen's ventricles; or lastly, that of strings of granules interposed betwixt the cartilaginous rings of the trachea. The glands themselves are encircled by a reddened areola, contain yellowish white mucus, and are softened—where the catarrh has been rather acute; otherwise they are hardened, of a grayish colour, and display a black speck, representing the more or less dilated duct. All these changes are most commonly met with at the posterior wall of the windpipe, in the mucous membrane of which they sometimes attain their highest pitch of development. Thus glands, either single or in groups, may enlarge so much as to form a tumour, which will project both externally and within the trachea, and attain the size of a pea or more. The glandular cyst is then found materially thickened, of a reddish or brownish-gray colour, and intimately associated with the adjacent, hardened cellular texture; the cavity of these cysts becomes likewise dilated, and filled with tenacious mucus, their ducts remaining patulous, and being surrounded with a reddened or blackish-gray areola.

The high degree of hypertrophy, constituting *diverticula* of the windpipe and bronchi, though rare, is nevertheless observable now and then, and is described by several authors (Morgagni, for example, Ep. xliv, 15); but its consequence, namely, the irregular expansion of the tracheal tube, was first pointed out by Rokitansky (Ueber divertikelähnliche Erweiterung der Luftröhre, Oest. Jahrb. N. F. vol. xvi, p. 374). Here the posterior wall of the trachea is found dilated into a pouch, the two horns of the cartilaginous rings being often parted, upwards of an inch; the mucous membrane is corrugated, and protrudes in a funnel or sac-like manner between the transverse muscular fibres, which, being thickened, form around each of the diverticula an elevated marginal rim. The elastic longitudinal fibres are for the most part attenuated and transposed, and, at the parts chiefly involved, found to have given way altogether. At the bottom of the funnel-shaped bulgings can be generally discovered the dilated duct of more or less hypertrophied glandular cysts, filled with mucus, which are sometimes lost by extreme distension, at other times constricted or cut off from the diverticulum by the transverse fibres. Rokitansky has met with this condition in six individuals of the male sex, of various ages, and in various stages of development; I have myself seen it, in its extreme degree, but once,—in a phthisical female. In this case, as well as in one of those described by Rokitansky, the same condition extended to one of the bronchial trunks. In the majority of the individuals affected, lingering or repeated catarrh had preceded death, which accrued from a variety of causes.

## SECT. II.—DILATATION OF THE BRONCHIAL TUBES.

## BRONCHIECTASIS.

This disease was first described by Laennec, and although of frequent occurrence, never adverted to by any prior writer. Whilst, however, we admit the importance of the facts first communicated by Laennec, our present experience of the subject obliges us, in some measure, to dissent from his theory respecting its pathology.

According to the above writer, three principal forms may be distinguished. The first exhibits a single point of one or another bronchial tube, mostly of the third or fourth order of magnitude, dilated into a cavity of a more or less regularly spherical shape. In the majority of cases the walls are at the same time attenuated, the cartilages and the fibrous texture being for the most part stretched asunder, if not made to disappear altogether, and the mucous membrane changed into a thin, smooth, almost transparent pellicle. In the second form, we find a *series* of these cystic dilatations occupying the course of one and the same bronchial tube, which in the intervals retains its natural width. The attributes of this form, and especially the manner in which it originates, prove it to be but a variety of the first form; for in neither instance does the dilatation constitute the extreme end of a bronchial tube, that is to say, a cul-de-sac, but either allows the cylinder to issue from it again, in its normal caliber, for further distribution, or else opens with small orifices into the lesser bronchia beyond. The third form is, however, quite distinct in character from the two previous ones. Here several bronchia, sometimes the majority, if not all those of an entire lobe, are uniformly dilated along their whole extent, making that portion of the lung appear to consist exclusively of tubes, widened to four or even eight times their natural size. The dilatation is not quite regularly cylindrical; the tubes generally increase in width towards their extremities, so as to give to a horizontal section of these bronchia, as they lie side by side, the appearance of the fingers of a glove. The dilatations constitute blind sacs, presenting neither a return to the normal dimensions of the tube, nor a continuation through smaller ones. The parietes of these dilated bronchia are never attenuated, as is usual in the other forms, but, on the contrary, thickened, the mucous membrane being turgescent, and furnished with folds, which resemble the valves of the small intestines, are more or less of a purple hue, and loosely encircled, without, by a thick, solid, yellowish-white fibro-cellular sheath. In certain instances the several varieties of bronchiectasis coexist in one and the same lung; and there are even examples of cylindrical, passing here and there directly into spherical dilatations, without deviating, in the character of their bronchial parietes, from the descriptions already given. On the other hand, the spherical dilatation is not invariably associated with attenuation of the walls, and especially of the mucous membrane. Hence it is seen, that in bronchiectasis, just as in other organic diseases, external form does not offer a marked and essential ground of distinction, independent of a careful estimate of the concomitant organic changes, and of their mode of development.

Laennec contented himself with ascribing bronchial dilatation to a mechanical and accidental cause, namely, the local accumulation of a tenacious mucus, and the efforts employed for its expulsion. It is not intelligible how even a single cavernous dilatation, much less how variety of form, could thus be occasioned,—to say nothing of the necessarily more frequent occurrence of the disease,—did it arise from so common and simple a cause. Andral assumes a hypertrophy of the bronchial canals originating in bronchitis, but omits to explain how this should cause the pulmonary substance to give way and become obliterated; for certain cases, indeed, he is driven to the opposite explanation, namely, that of the bronchi being atrophied. A more plausible theory, to which we shall afterwards revert, was first assigned by Stokes. In all these explanations, the air-passages are represented as the part originally affected, whilst the remainder of the lung is regarded as but passively implicated. Of recent years, however, the pulmonary cells themselves have, in many instances of bronchial dilatation, been looked upon as the part primarily diseased, and the bronchi as secondarily affected. In this sense a remarkable hypothesis has emanated from Corrigan, (Dublin Journ. May, 1838, p. 266.) He compares the condition of the pulmonary texture, in bronchiectasis, with that of cirrhosis of the liver, assuming a fibro-cellular texture to become developed, whether through inflammation or other causes, within the lung,—the normal longitudinal fibres of the bronchial walls to unite therewith, and produce constriction, and ultimately atrophy and obliteration, of the pulmonary cells. Hereupon the bronchial tubes would seek through excessive dilatation, to fill up the vacated space; at the same time the healthy lung, the heart, and the abdominal viscera, would be drawn over to the diseased side, and the thoracic parietes collapse proportionately. He terms the disease *cirrhosis of the lung.*

Dilatation of the bronchi is always a consecutive affection, and accompanied by other organic changes, denoting a disease, the course (not the consequences,) of which has terminated. It is for the most part of tardy development, and seldom attains that high pitch, at which it may eventually prove fatal, either through the embarrassed state of the respiration, or through its influence over other organs and systems. Generally speaking, it persists through, and when superadded to other accidental disorders, tends to shorten life. Bronchial dilatation would appear to be engendered by bronchitis, by tubercular disease of the lungs, and by pleuro-pneumonia, the two former being especially prone to produce the spherical, the last the cylindrical form.

Bronchitis exercises a more prompt influence than the other two diseases, and probably in the following way.—First, the air-passages are stripped of their epithelium-lining in the ordinary manner, their canals becoming loaded in part with a mucous secretion, in part plugged with fibrinous exudation. This latter occurrence takes place chiefly within certain of the lesser twigs, occasioning a collapse of the adjunct air-cells. The space thus set free is sought to be filled up by expansion of the neighbouring parts, giving rise in the majority of cases to emphysema; where, however, the collapse does not occur close beneath the surface of the lung, but at a greater depth and near a larger bronchial tube, and where it comprehends a larger tract of pulmonary substance, the result is bronchiectasis. These circumstances do not, however, suffice for the formation of a bronchial cavity; the parietes of the involved bronchial tube must needs have previously suffered the changes pointed out by Stokes, namely, loss, through inflammation, of elasticity in the longitudinal, and of contractile power in the annular fibres, with consequent incapacity on the part of either to resist the mechanical influence of forcible inspiration, or of violent cough. It is difficult to say whether Stokes is right in believing that a saccular protrusion of the mucous membrane is caused by yielding of the fibres;—such, however, may probably be the case, where the dilatation is one-sided, and its principal portion external to the axis of the bronchial tube. The analogy, likewise adverted to by Stokes, with the forms of aneurism, must, on the other hand, fall to the ground, as untenable. (See Aneurism.)

The bronchial cavities often met with in children, after hooping-cough,[1] and indeed all those found scattered either singly or in groups, at various parts of one or both lungs, are of similar origin. The mucous membrane, under such circumstances, exhibits, generally, the same characters, although by the recurrence of catarrh or inflammation, it may have become thickened, puffy, rugged, and more or less the seat of a thick, mucous, or purulent secretion. Bronchiectasis resulting from simple bronchitis, is accompanied with the fewest changes of other kinds in the respiratory organs,—for the most part only by the sequelæ of chronic catarrh, emphysema of the lung, &c. In the immediate vicinity of the dilated portions, however, the pulmonary texture is almost invariably impermeable and lax, or it may consist of nothing beyond the apparent remains of cellular tissue.

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[1] Whether Guersent is right in conjecturing bronchiectasis to be sometimes congenital, remains to be proved. It is scarcely probable.

Tubercular disease of the lungs, both during, and after completion of the healing process, constitutes one of the most frequent causes of several of the less sharply-defined forms of bronchial dilatation. Reserving for a subsequent chapter a more enlarged consideration of the subject, we shall here confine ourselves to briefly stating that which relates to the bronchi. In these cases there is considerable corrugation of the portions of the lung altered by tubercle, and the bronchi, like the other parts, are engaged in compensating for vacant spaces within the thorax. Hence the apex and the upper half, generally, being the first seat of tubercular mischief, are for that reason the almost invariable locality of bronchiectasis. In following the ramifications of the air-passages through the upper lobe of the lung, whose apex has become shrivelled from tubercular disease, we frequently discover individual branches displaying, along the whole of their course, often to the extent of an inch or more, a caliber much enlarged, in proportion to that of the branches from which they emanate. They traverse a texture, in a great measure, if not wholly, impervious to air, and mostly reduced to a somewhat hard, cellulo-fibrous mass. The dilatation gradually increases towards the termination of the altered tube, which constitutes a blind sac; all the ulterior branchlets having, as shown by a section of the lung, undergone obliteration. The mucous membrane of such a tube is plicate, and throws out valve-like processes, but remains otherwise smooth. The lateral branches are in like manner obliterated, individual branchlets only passing here and there into a group of still permeable air-cells. At another time, the entire apex of a lung is, to a certain depth, converted into an almost cartilaginous mass, wherein the bronchi have all suffered obliteration, and are cognizable only as white, thread-like ramifications.

Below, where these changes terminate, the larger branches implicated are found dilated into irregular, jagged cavities, with a thin and smooth mucous membrane within, and a thick cellulo-fibrous coating without. In other cases again, bronchial tubes which pursue a regular course up to a given point, suddenly dilate into spindle-shaped cavities, and beyond these are thoroughly obliterated; the mucous membrane appears rough and decayed, and the cavity itself replete either with thick mucus, or with a more or less consistent, tubercle-like mass. The avenues to these spindle-shaped dilatations are often greatly contracted.

Lastly, there occur in lungs, previously affected with tubercular destruction several conditions liable to be mistaken for bronchiectasis. Such are instances of bronchial tubes leading into tubercular cavities still open, but modified by the healing process. At times a large branch passes, with a funnel-shaped dilatation of its orifice, directly into such a cavern, its mucous membrane merging insensibly into the lining membrane of the latter, so as to render it difficult to distinguish their respective limits. At other times the branch leading to the cavity becomes progressively narrower, and, at the threshold, strictured if not thoroughly obliterated, so as to convert the original vomica, furnished, in such cases, with a serous or mucous lining membrane, into a shut sac. At other times, again, two or more, mostly of the larger branches, are found to have united, at the centre of shrivelled texture, to form an irregular flat cavity, which would seem to be a continuation of those tubes, having, like them, a rough and eroded lining membrane. In all these cases the phenomena indicative of the cure of tubercular disease, are abundantly present.[1]

The most remarkable species of bronchiectasis is that which results from extensive obliteration of the pulmonary cells, induced, in all probability, through pleurisy and pneumonia. This is the *third* form above designated. I have met with it, most frequently, in the inferior lobe, especially of the left lung, where it occupied the posterior external portion,—in one instance, two thirds of the entire lobe. Twice I met with it in the middle lobe, and once in the anterior half of the upper lobe of the left lung. Not only the parts implicated, but likewise the greater portion of the entire lung, invariably coalesced so firmly with the costal pleura, as not to admit of being separated without difficulty, or partial rupture. In several of the cases there were vestiges of a by-gone violent and inveterate pleurisy, in the form, partly of thick and more or less organized remains of plastic exudation, partly of calcareous plates. The proper pulmonary cells were transformed into a pale reddish, or pale brownish, flabby, soft mass, utterly impervious to the air, and not susceptible of inflation. On cutting into this texture the bronchia were seen in close juxtaposition, and filled with a thick yellowish mucus, like so many elongated abscesses; in most cases the diseased lobes had diminished in volume; in two instances only did they occupy a greatly enlarged space. In all the examples of cylindrical bronchiectasis adduced by Laennec and Andral, the same relations prevailed. Several grades of this morbid condition have been clearly distinguished. In one case the obliteration did not extend to all the pulmonary cells pertaining to the dilated bronchia; here the dilatations did not all constitute blind sacs; some sent off small twigs of normal caliber, and capable of inflation, whilst everywhere else the impermeability was complete. When, on the contrary, the disease had attained a very high degree of intensity, the texture of the lung was completely transformed, so that not a vestige either of pulmonary cells, or of the lax intermediate texture before described, was any longer perceptible, the whole space being taken up by the dilated bronchial tubes, which, at their extreme end, widened into sacs, separated only by their walls. I have never, as some affirm to have done, found these walls so far destroyed by atrophy, as to establish common multicellular cavities. Careful inflation and cautious incision invariably showed them to be separate. This form of bronchiectasis arises, in all probability, where the pulmonary cells have become obliterated by previous pneumonia, and the bronchial tubes been constrained by the pressure of the air to fill up the space vacated, before the parietes of the thorax have had sufficient time to collapse. *Pleurisy* may, in like manner, give rise to dilatation, where the effusion is of a character to keep the pulmonary cells long compressed, without subsequently affording them an opportunity for due expansion. (See Pleurisy.) The bronchial tubes, not being similarly encumbered, are the more liable to yield to the pressure of the air inspired, from the comparatively tardy collapsing of the thoracic walls. This passive dilatation will of course be promoted, where the parietes of the air-passages have been deprived of their elasticity, by the aforesaid inflammatory influences.

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[1] Most of the changes alluded to are carefully defined and figured in Reynaud's essay on Obliteration of the Bronchia. (See Mém. de l'Acad. Roy. de Méd. vol. iv, 1838.)

Most forms of bronchiectasis, being accompanied by an alteration in the volume of the lung, a corresponding displacement of the surrounding parts must needs follow. This is least manifest in the simple cavernous dilatation resulting from bronchitis,—a form that seldom attains a great height, and occurs singly at various parts of the lung, unattended by any remarkable collapse of the air-cells. The dilatations arising from tubercular disease, generally occupy only the apex of the lung; the subsidence of the thorax is therefore confined to parts corresponding. It is, however, rather due to the shrivelling of the pulmonary substance, occasioned by the cicatrization of degenerated tubercle, and, in point of fact, the sinking in question would be prevented by the bronchial dilatation, were the latter proportionately developed. The cylindrical form of bronchiectasis, just described as consequent upon extensive obliteration of the pulmonary substance, is on the contrary almost always attended with a marked shrinking of the parietes of the thorax in the corresponding situations, and, in strongly developed cases, produces, at the same time, a remarkable displacement of the viscera. Corrigan's second case (l. c.) furnishes a notable example of this last-mentioned occurrence. The right lung had become greatly diminished in size; the left, on the contrary, enlarged, bulging over towards the right; the heart lay almost entirely on the right side, and the diaphragm and liver were more than usually elevated. This form of bronchial dilatation is, however, not always accompanied by a diminution of volume; on the contrary, where the dilatation attains its utmost development, the diseased portion occupies more space, leaning over towards the opposite side, and producing a greater amount of vaulting of the thoracic parietes. Thus I found it, in the aforesaid case of extensive bronchiectasis, in the anterior half of the superior lobe of the left lung.

Where bronchiectasis is associated with impermeability of a large portion of the parenchyma of the lung, the impaired circulation gradually produces an accumulation of venous blood within the veins generally, and within the right heart in particular.[1] Hence, in progress of time, a mostly passive dilatation of the right ventricle and auricle, and, occasionally, where the organism has sufficient energy to oppose a forcible reaction to the existing impediments, hypertrophy of the walls of the right ventricle.

Where, however, the condition in question is of very long standing, and the vital powers unequal to the struggle, the general consequences of impeded circulation become, ere long, manifest. *Dropsical* phenomena eventually set in, and extinguish life; if, indeed, complications of another kind do not close the scene at an earlier period.

Certain relations between bronchiectasis and tubercular disease, still remain to be studied. We have seen above that the cure of phthisis is often brought about, partly through the dilating of bronchial tubes: it is therefore the more remarkable that the cylindrical dilatation of the bronchi, last described, never coexists with active tubercular disease, and rarely even with decided traces of the extinct affection. It is not hereby implied that the two diseases are necessarily incompatible with each other, but merely that bronchiectasis determines in the organism certain conditions unfavorable to the further development of tubercles. This remark applies chiefly to extensive cylindrical bronchiectasis; that which results from the cicatrizing of tubercular cavities, being nothing more than an accompaniment, a subordinate consequence of the healing process, which latter is due to other causes. Nor does it, as certain cases indicate, offer any barrier to the renewal of active tubercular disease. In like manner the cavernous form, which has its source in bronchitis, cannot be looked upon as essentially opposed to tubercular disease, as is proved by the well-related example of J. F. H. Albers (Beobacht. auf dem Gebiete der Pathol, vol. iii, p. 82.)

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[1] This venous stagnancy, in some instances, betrays itself during life, by more or less violent attacks of hæmoptysis.

## CHAPTER IX.

### EMPHYSEMA OF THE LUNG.

The term "emphysema," though not altogether appropriate, is, nevertheless, sanctioned by Laennec. It designates, first, a morbid dilatation of the pulmonary cells, (vesicular emphysema); and, secondly, an extravasation of air into the cellular texture of the lungs, and beneath the pulmonary pleura (interlobular emphysema.)

*Vesicular emphysema* is exceedingly frequent at every age, but more especially at an advanced period of life. An experienced observer will, on minutely examining a lung, seldom fail to find some of its vesicles dilated.[1] But the mischief is, for the most part, too slight to reveal itself by any obvious symptoms during life, and may easily escape notice on cadaveric inspection. In many cases emphysema attains such a pitch as to occasion symptoms of the most striking and distressing nature, and likewise to lead to, and materially influence the course of other diseases. The following account of the general changes wrought by it upon the structure and relations of the respiratory, and of a portion of the circulating organs, will allow a correct judgment to be formed with respect to the consequent functional disturbance.

Laennec, who first pointed attention to the disease, assigned catarrh as its cause, and more particularly that species, which he somewhat illogically termed "dry catarrh." The majority of later writers on pulmonary diseases, have adopted this view, and have sought to corroborate it by the results of their experience. Louis, whose researches on this point are most elaborate, alone dissents from the general opinion.[2]

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[1] In 53 cases of cholera, 23 of the individuals were found by Louis to be more or less affected with emphysema of the lungs, in various stages. Nor was there any ground for the assumption that it had become developed during the fatal malady.

[2] Mém. de la Soc. Méd. d'Observ. p. 160. Paris, 1837.

My own observation of the entire course, the duration, and the general character of the disease, induces me, to a certain extent, to agree with the majority. Catarrh is, without doubt, the principal occasional cause of emphysema; and hooping-cough, in particular, is evidently capable, within a very short space of time, of effecting the highest degree of dilatation in the pulmonary cells. The character and real import of emphysema must, however, be admitted to depend on other causes, as yet not thoroughly made out.

With reference to the hereditary nature of emphysema, the experience of Jackson (upon whose deductions Louis's treatise is partly founded) is important. He remarked, namely, that out of 28 cases of confirmed emphysema, 18 appeared to be hereditary, one or other parent having, in all of them, been similarly affected. Of 14 of the individuals, in whom the disease set in very early in life, the parents were well known to have been afflicted with asthma.[1] In two instances the emphysema was the immediate effect of strong mental emotion of a depressive tendency, independent of any other obvious cause. It is, on the whole, surprising how rapidly dilatation of the pulmonary cells may take place,[2] although it requires time to attain such a height as to manifest itself by physical signs. The means whereby catarrh effects a dilatation of the cells, are purely mechanical. In catarrh, the lesser bronchial twigs are obstructed, partly by tenacious mucus, and partly by the swollen mucous membrane. During inspiration, the action of the muscles is powerful enough to impel the air through these obstacles: expiration, which is carried on rather through the force of elasticity than through the medium of the comparatively feeble expiratory muscles, is less complete. Thus more air is inhaled than exhaled, and the surplus, expanding through the natural warmth, ultimately overcomes the resistance due to the elasticity of the pulmonary texture. On the other hand, the more forcible acts of expiration—coughing for example—will rather compel the surrounding vesicles to yield to the pressure of the dilated ones, than allow the air, contained within the latter, to surmount the obstacle to its natural exit. In like manner, swellings of the bronchial glands, &c., or tubercular deposits within the lungs, may give rise to emphysema, by the compression of bronchial tubes,—as shown by Reynaud, Andral, Carswell, and others. To enlarge still further upon the catarrhal mode of development, would involve the repetition of much that has been already said under the heads of Catarrh and Bronchiectasis;—to these the reader is accordingly referred.

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[1] On the other hand, out of 50 individuals entirely free from emphysema, the parents of three only appear to have been affected with the disease.

[2] This is evident from Louis's sixth case, and also from an analogous one which came under my own observation. In a scrofulous child, of ten years, whose breathing had never previously been affected, an attack of scarlatina was followed by the ordinary signs denoting tubercle, and, within a fortnight, death ensued. In both lungs were found yellowish and very soft miliary tubercles, and, at the intermediate points, the pulmonary cells were considerably enlarged,—many of them appearing as big as grains of hemp seed. The substance of the lungs was replete, throughout, with blood and serum.

*Do the pulmonary cells undergo hypertrophy, or atrophy?* This question has occupied the attention of many pathologists, and especially of Andral and Lombard. It would be fraught with less of practical interest, were it not for the common habit of connecting the idea of hypertrophy and atrophy with certain therapeutical deductions, altogether inapplicable to the present disease. Schönlein has of late restricted and more accurately defined the meaning of the above terms, and, in unison with his views, they would certainly not apply to positive disorganization, like emphysema. In an anatomical point of view, it would, however, still be interesting to determine, whether dilatation of the pulmonary vesicles is attended with an increase, or with a decrease of organic substance,—with thickening, or with attenuation of the walls of the cells. In the majority of cases, indeed, this is scarcely possible. To settle the point, Louis suggests taking one piece of emphysematous and another of healthy lung, pressing out every particle of air from both, and then ascertaining, whether the emphysematous portion be the thicker or the thinner of the two. The difficulty, nay, impossibility of conducting this experiment with accuracy, must be obvious to every one, and it is hardly conceivable how so careful and exact an observer as Louis could gravely hazard such a proposition.—It is convenient, for general reasons as well as in relation to the point at issue, to class dilatation of the pulmonary cells under three heads. The first variety is merely to be regarded as a change incidental to senescence, and might be deemed simply physiological, were it not, like every other organic relation belonging to old age, associated with impairment of the natural functions. It has been most fully described by Hourmann and Dechambre, who instituted comparative researches on the structure of the parenchyma of the lungs, at each period of life.[1] On opening the thorax of old persons, even where no serious disturbance of respiration has been noticed, the lungs appear greatly shrunken,—distending the cavity far less perfectly than in young individuals. These organs are flabby, and preternaturally dry; deficient in elasticity; not audibly crepitant; soft and woolly to the feel. In most instances they contain a considerable proportion of black pigment. This state of things is due to marked wasting of the walls of the vesicles; thus the condition closely borders upon genuine atrophy. Hourmann and Dechambre have represented three stages as typical of this wasting. In the first the volume of the lung is as yet undiminished; but the cells are more capacious, and their walls attenuated, whilst the division into lobules is still conspicuous. Presently, however, the interlobular cellular tissue becomes here and there deficient, and inadequate to keep up the distinction of the lobules. Meanwhile a coalition of cells has been effected at intervals through the effacement of their walls; although, from the equable nature of this process, the cells have almost all assumed an oval shape. At length the division into lobules ceases to be discernible; the enlargement of the cells is now very considerable, though unequal and irregular, owing to the union of clusters of contiguous vesicles into single cells, whose irregularity is further increased by bands or filaments, the remnants of former septa. Throughout the lungs, the number of capillary blood-vessels is naturally diminished, and the parietes of the cells even appear much less vascular than usual. In very advanced age, all the bronchial canals, and, more especially in males, the trachea and larynx are in like manner found dilated.

The second variety of pulmonary emphysema is represented by extensive dilatation of single vesicles or of small insulated groups of vesicles, without material implication of the rest of the lung. At the apex or edges of the lung, more especially the anterior edge, are seen projecting single vesicles of different sizes, and sometimes of a perfectly regular spherical shape, though for the most part of uncertain figure, many of them being oval, like the swimming bladder of fishes. Their parietes are in general extremely attenuated, almost to transparency, and exhibit but few, delicate, vascular ramifications. When grouped, these vesicles either rest upon a broad base, or else penetrate deeply into the healthy parenchyma of the lung: when single and regularly shaped, they stand out upon pedicles. These solitary vesicles vary considerably in magnitude, attaining sometimes that of a small apple. On being punctured, in the recent subject, they fall away to nothing. When opened, after the lung has been dried, all the larger ones, and those of irregular shape, bear traces of the coalition and blending of several pulmonary vesicles into one, the base and the sides of the cell showing distinctly the remains of several vesicular septa. At their base, there are at the same time discoverable sundry orifices in communication with the adjacent bronchial twigs. The dilatation of pulmonary vesicles is not necessarily the result of the effacement of their walls, or of the union of several cells, but may arise simply from excessive distension of the parietes,—a species of degeneration altogether peculiar, and necessarily distinct both from hypertrophy and atrophy. Where this degeneration is limited to a few vesicles, leaving the rest of the lung intact (as I have frequently seen), it induces no very marked symptoms during life, and hardly deserves the name of a disease. Occasionally, however, the entire surface of one, or of both lungs is closely beset with dilated vesicles, either solitary or aggregated; under such circumstances, the patient is afflicted, for years, with the most grievous sufferings, and eventually succumbs to the malady. This likewise happens where this form occurs in union with the very common one about to be described.

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[1] Archives générales de Médecine, Août 1835.

Excessive distension may terminate in rupture of the emphysematous vesicles, and this rupture, in the absence of pleural adhesions, give rise to *pneumothorax*, which, without causing inflammation, may compress the lung in such wise as to destroy life.[1]

The third species, which, for distinction's sake, I would term lobular vesicular emphysema, is the structural change upon which most commonly depends the train of symptoms recognized since Laennec's time as emphysema. It affects one lung as often as the other; more frequently both at the same time. The upper half of the organ appears much more prone to the affection than the remainder: for in those of Louis' cases, where the emphysema did not extend over the entire lung, the superior lobe was involved in sixteen, the superior and middle lobes in three, the middle lobe in one, and the inferior in five only. Again, this form assails the front in preference to the hinder surface of the lung,—the vesicles most dilated, being always observable at the anterior sharp edge. Although the disease may not involve the whole of a lobe, yet it invariably seizes upon more or less diffuse groups of vesicles, which frequently all become dilated to a uniform degree. These groups seem raised above the rest of the surface of the lung: for, whilst the sound portions collapse somewhat on the thorax being opened, the diseased ones, devoid of elasticity, remain filled with air, so as to resist the atmospheric pressure. They are paler, and by their whitish, pellucid look, often contrast strikingly with the healthy textures. The single cells are easily recognized, being often as large as grains of hempseed; collectively, they represent a tuft-like elevation, or supernumerary lobule. Sometimes, in front, where the two borders of a lobe conjoin, a lobule is found dilated, in such wise, that it hangs down one or more inches, like a long, thin appendix. Here there is invariably a mutual blending of the single cells, as may be ascertained either by a simple puncture made at any one point, which will cause the whole appendix suddenly to collapse, or else by drying the part, and then cutting it asunder,[1] when it will be found to contain several empty cavities, of irregular form and unequal size, the septa of which are either riddled with holes, or reduced to narrow bands, if not to mere shreds. It is a remarkable fact that the cells of such a lobule increase in width, progressively, from the centre to the periphery. An emphysematous portion of lung is soft and woolly to the feel, offering the same gentle resistance as an eider-down cushion. On moderate pressure it crepitates very feebly, if at all;—on the pressure being increased, some large but rare crepitation may be perceived.

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[1] See a case by Stokes (Dubl. Journ. Sept. 1840. p. 151.)

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[1] The drying of emphysematous pieces of lung shows plainly how little air can escape from dilated cells; even in preparations kept in alcohol, the diseased cells retain their air, and continue to do so when the preparation is slowly drying,—whilst the undilated cells dwindle down into a hard and almost solid mass.

The dilated pulmonary textures appear, when cut into, whitish, or of a pale vermilion, somewhat like the lungs of a calf. They are preternaturally dry, and their cut surface uneven, owing to the collapse of the different-sized vesicles corresponding to it. When the healthy portion of the lung is incised, the exsanguineous condition of the emphysematous, offers a striking contrast; for the diminution of the capillaries is evidently *absolute*, not merely relative to the augmented space. It is easy to trace, on the cut surface of a dried portion of lung, the confines of lobules, whose vesicles are more or less dilated.

Where the malady is not confined to individual lobules, or lobes, but affects, as is more usual, the greater portion of one or both lungs, these organs mostly press forward, the instant the thorax is opened, as though cramped for space; and their anterior edges touch, or even overlap each other; the individual lobes are pushed up in such wise that their angles and edges strive, in every direction, to find room. The anterior margin of the left lung almost completely covers the heart, burying it deeply within the chest. The lungs, on being taken out, do not collapse, even after separation of the individual lobes. Such are the features which distinguish emphysema, as a disease, from the mere effects of senile atrophy.

This constant repletion and distension of the lungs with air, just as after the fullest inspiration, produces, in the course of time, a very marked influence upon the shape of the thorax, and upon the position of neighbouring parts. The whole chest acquires a rounder, more arched shape, especially between the clavicles and nipples. This vaulting closely corresponds to the seat and amount of the malady, and therefore occupies one side or the other, (according to Louis, more frequently the left,) or is limited to certain portions of the chest only. It is more perceptible in front than behind, owing to the peculiar movements and direction of the ribs, and likewise to the emphysema itself affecting, as already stated, a preference for the anterior surface of the lung. In aggravated cases, however, I have occasionally seen the projection behind marked by the relative position of the shoulder-blades, which stand out like a plane forming a tangent to a convex surface. At the abdomen, the false ribs start forward on either side. Louis mentions another kind of projection, or rather filling up, of the supra-clavicular region, which gives a plumpness, he says, to the otherwise emaciated neck. I believe this appearance to have been present in the cases noted by that accurate observer, but have never myself witnessed it. Indeed, I have mostly found, above the vaulting of the chest, and above the clavicles, a decided pit or depression, wherein are displayed, almost like thick, tense cords, the cervical muscles of inspiration, which in general play a subordinate part, but here, where the operation of the principal inspiratory muscles is defective, become prominently active.[1]

Despite this permanent expansion of the walls of the thorax, and the extraordinary breadth of the intercostal spaces, the intercostal muscles still continue to act, so that the interspaces are rendered conspicuous by a deep furrow between the prominent ribs,—a feature pointed out by Stokes as distinguishing emphysematous dilatation from that produced by empyema and similar affections (see Dubl. Journ. vol. ix, No. 25, March 1836). Under these circumstances, the ribs themselves remain all but motionless, and in most instances the cartilages ossify in course of time.

The lungs occasionally gain space, by forcing down the diaphragm deep into the abdomen, and permanently establishing its convexity in that direction. Like the intercostal muscles, this muscle does not cease to act, as Stokes has ably shown, but still goes on making the same efforts, though unsuccessfully, to bring about sufficiently deep inspirations. It is remarkable that, during inspiration, the abdomen bulges out in a hemispherical shape, while the false ribs are at the same time drawn inwards.

The several viscera of the abdomen are displaced, together with the diaphragm; the liver and spleen, in particular, are pressed downwards. The stomach, indeed, commonly retains its position, but being prevented from fully expanding, is found diminutive and contracted, which accounts for the distressed breathing, caused by undue distension of the stomach with food or gas, in emphysematous patients.

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[1] The circumstance of every auxiliary muscle of inspiration being roused to constant and vigorous action, gives to emphysematous patients a peculiar expression of discomfort or embarrassment. The labour in breathing is at once apparent: the alæ nasi are drawn widely asunder,—the angles of the mouth downward. The shoulders are alternately elevated and depressed,—the cervical and abdominal muscles are kept in brisk action, whilst the thoracic muscles of inspiration are seemingly quite inert,—the thorax constantly maintaining that degree of expansion which, in health, only accompanies the deepest inspiration.

The heart is in like manner liable to become displaced, not only towards the spinal column, but also downwards, frequently to the ninth or tenth rib, and very commonly towards the epigastrium. The inevitable consequence is great embarrassment to the circulation. Where the emphysema is altogether, or for the most part, limited to one lung, the latter may increase in volume so far as to force the heart over to the opposite side,—a circumstance sometimes palpable during life.

The dilatation of air-cells is, however, not productive merely of mechanical mischief, but also of secondary changes, naturally, so to speak, resulting from the disease. Foremost among these are hypertrophy and dilatation of the heart, the development of which, during the progress of emphysema, is generally admitted. The affection has its seat, for the most part, in the right ventricle and auricle, though occasionally in the left heart also. Louis met with this complication in 10 cases, out of 19 in which the lungs were more or less emphysematous; in extensive emphysema, I have only once found it wanting. In true senile atrophy it is, on the contrary, rare. The connexion between the disease of the central organ of circulation and that of the lungs, can be distinctly traced, both diseases being cognizable, with tolerable certainty, during life. The former affection is the consequence of the latter; for in all the cases observed by Louis, the palpitations, &c. were preceded, during a longer or shorter interval, by troubled breathing. It may be readily conceived how greatly the vital phenomena must become modified by such a complication,—one requiring perhaps, for its full maturation, a period of twenty years. Several circumstances concur to induce dilatation and hypertrophy on the right side of the heart; of these the most influential is the disturbance to the respiratory function. The elasticity of the pulmonary texture being impaired, and inspiration at the same time defective, aeration falls very far short of its natural standard; while, again, the anæmious condition of the emphysematous tissues prevents the lungs from taking up more than a limited amount of blood. Hence arises an impediment to the venous circulation, and a consequent stagnation of venous blood in the right side of the heart, in which situation, owing to the preternatural efforts of the entire organ to support the obstacle, passive dilatation ultimately ensues, notwithstanding the thickening of the walls; at the same time, the larger venous trunks, and organs abounding in veins, such as the liver, become surcharged with blood. Hence the complexion frequently becomes cyanotic, and jaundice, of greater or less intensity, sets in. To this may be superadded the unnatural position of the heart, of itself an obstacle to the circulation. Stokes annexes another circumstance, which may occasionally have some weight. It is this, that, whenever the heart is forced down far into the epigastric region, being compressed on the one hand by the dilated lungs, on the other by the abdominal organs, it is thus deprived, in part at least, of the protection of the thoracic wall, and compelled to redouble its efforts for the due performance of its function, whereby an augmentation of its volume is brought about. In this case alone the left ventricle becomes simultaneously hypertrophied; under the ordinary circumstances it has rather the appearance of being atrophied, its walls being thin, at least when compared with those of the right ventricle.

At an advanced period of the disease, and especially when the consecutive heart affection has become established, the tardy venous circulation frequently operates as a source of dropsical accumulations, which appear first in the shape of œdema of the lower limbs, but sooner or later in that of general dropsy.

It has been before stated, that Louis questions the influence of catarrh in causing emphysema; but he even denies its importance as a complication. Few pathologists will be disposed to concur in this opinion. My own experience convinces me that no disease supervenes upon emphysema more commonly, or upon slighter occasions, than catarrh. It is often, however, more easily got rid of by emphysematous patients, simply because, as it proves doubly distressing to them, it is nursed with greater care. Louis mentions, as one reason for assigning to catarrh little influence in producing emphysema, that he has seldom found the latter disease to coexist with dilated bronchia. But it has been already seen that bronchiectasis is very frequently dependent upon other causes besides catarrh;—nor have I found its coexistence with emphysema to be, relatively speaking, uncommon.

The mutual relations between emphysema and tubercular affection of the lungs, merit special consideration, as being calculated to throw light upon some of the causes of the former disease, hitherto but imperfectly understood. Those much in the habit of examining the dead body, cannot but be struck with two circumstances: first, the almost invariable existence of emphysema in lungs which bear the characteristic marks of recovery from phthisis,—and, secondly, the proportionate rareness of tubercular deposits in emphysematous portions of lung. This would seem to show that dilatation of the air-cells constitutes one of the conditions under which the cure of phthisis is possible, and again that it forms an obstacle to the development and progress of tubercle. Those facts have been so amply confirmed by observation, as to lead to the assumption, that a high degree of emphysema will almost preclude tubercular disease, although a slighter degree and limited extension thereof may fail, either permanently to arrest the progress of pulmonary consumption, or to hinder fresh tuberculous deposition. The manner in which the air-cells become dilated, during the cicatrization and collapse of tubercular portions of a lung, furnishes at once a physical explanation of many cases of emphysema. Where the destruction of an extensive portion of lung is followed by absorption of the liquefied portions of the tubercle, shrivelling and cicatrization of the cavities, obliteration of blood-vessels, bronchia, and pulmonary cells,—in a word, by the healing process,—a considerable space is thereby vacated within the implicated pleural cavity. This can be filled up only very imperfectly, and very gradually, by a sinking in of the walls of the chest, whereas the dilating of the air-cells in the still permeable textures of the lung, affords a far readier medium of compensation for the disparity between the capacity of the cavity, and the volume of the contained organ. Hence it has been often observed, after the cure of phthisis is accomplished, that the parietes of the thorax not only do not shrink, but even rise, immediately below the clavicle, in the vaulted manner peculiar to emphysema. It need scarcely be pointed out that the abiding cough and increased necessity for inspiring tend greatly to promote the incipient dilatation of the air-cells.

The different forms of active hyperæmia are likewise subject to the control of emphysema. Emphysematous portions of lung seldom become affected with œdema. *Pneumonia,* for the most part, leaves exempt such lobules as happen to be emphysematous; in some instances, however, whilst the non-emphysematous textures become thoroughly hepatized, by means of fibrinous exudation, those of which the cells are dilated, fill either with a thin, a tenacious and ropy, or a gelatinous mass. I have only once met with an apoplectic clot in the emphysematous parenchyma of a lung.

*Interlobular emphysema of the lung* results from the escape of air, out of ruptured vesicles, into the cellular texture of the lung. According to Laennec, the air may pass from the injured part to the root of the lung, from thence into the mediastinum, and even into the subcutaneous cellular tissue of the neck. This is, however, probably a rare occurrence, the small amount of cellular tissue, within the lungs, counteracting much accumulation, or the easy and rapid diffusion, of air. On this account the extravasated air is chiefly found beneath the pleura and around individual lobules. In the former situation it is sometimes seen in transparent, moveable vesicles of various magnitude. Between the lobules it forms into parallel, and more or less narrow strata, linked together by transverse strata, as accurately figured by Carswell, (Fasc. ix, Pl. 1, fig. 7.) By this extravasated air, the pulmonary cells are proportionately compressed. Interlobular emphysema is referrible to the rupture of one or several pulmonary vesicles, for the most part through violent exertion in lifting, straining, shouting, or coughing. Laennec believed the rupture of *dilated* cells to be the cause. It is, however, to be remembered, that in vesicular emphysema the cellular tissue of the lungs is already in a high degree attenuate, and therefore in a condition by so much the less favorable for development or expansion. Upon the whole, interlobular emphysema appears to be a disease of subordinate import, and of rare occurrence, for which reasons, perhaps, it has not been made the subject of very minute research. I have never seen it before death, or detected its traces afterwards, except in subjects where rapid decomposition had caused the formation of gases within the cellular tissue of various other organs.

# CHAPTER X.

## TUBERCULAR DISEASE.

### SECT. I.—TUBERCULOSIS OF THE LUNG—PULMONARY PHTHISIS.

Of all organs, the lungs are by far the most prone to become affected with tubercle, nor are they by any means wont to remain exempt, even where it is originally deposited elsewhere. The production of tubercle takes place under circumstances the most varied, and occupies a longer or shorter time; it advances either with steady progression, or remains stationary at some point of development, for a shorter or longer period, proportionate to age, constitution, complications, and supervening relations. The presence of tubercles never fails to cause abiding disturbance in the function of the portion of lung affected, whilst their elimination, for the most part, leads to destruction of the parenchyma of the lung (phthisis), and now and then only, in the long run, to imperfect recovery.

Few objects of pathological anatomy have been submitted to more minute research, by distinguished observers, than tubercle, and the most discordant views have been, and, to a certain extent, are still entertained respecting its nature, its origin, and subsequent destruction, its seat and structure, and, finally, its different forms. It would be foreign to our present purpose, and would, at the same time, far exceed our limits, to enter into the details of those divers theories, a task better suited to a work on *general* pathological anatomy. We shall refer only to the best sources of information upon those points,[1] and merely touch upon the *general* relations of tubercle, in so far as it concerns disease of the lungs, where it occurs not alone more frequently, but also in greater variety of form, than in any other organs, not excepting even the lymphatic glands. Although tubercular disease assails the respiratory organs at every age, it is most apt to invade them, in its utmost intensity, at the period at which the lungs have acquired their fullest activity,—namely, between the 18th and the 36th years.[1] We shall therefore chiefly investigate the anatomical history of phthisis during this period, and afterwards point out such deviations as characterize it in childhood, in manhood's prime, and in old age. Of the sexes, the female would appear to be the more prone to the disease.

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[1] Besides the writings of Laennec, Louis, Andral, Cruveilhier, and others, we would mention Carswell's 'Illustrations,' &c. Fasc. Tubercle; Cerutti's 'Collectanea quædam de Phthisi Pulmonum,' &c. Lipsiæ, 1839; Sir James Clark's work on 'Pulmonary Consumption;' Schroeder van der Kolk's 'Observationes Anatomico-pathologicæ,' &c. Fasc. i. Amstelod. 1826; and, lastly, Sebastian's treatise 'De origine, incremento et exitu Phthiseos Pulm. obs. Anat.' Groning. 1837.

It will be seen from the sequel, that the production of tubercle is not dependent upon accidental conditions,—upon some occasional cause possible under all circumstances, as for example—inflammation. We must, therefore, assume a general predisposition, and this would appear to be no other than scrofula. It would be irrelevant to endeavour to trace the general connexion between scrofula and tubercular disease; the only point for consideration is, whether or not a decided predisposition to tubercular disease be anatomically demonstrable.

A frame of body certainly indicative of future phthisis cannot be said to exist; most of the signs, as far as the thorax is concerned, betraying, not the predisposition to, but the actual presence of, tubercles in the lungs. This applies more especially to the projecting of the clavicles and shoulder-blades, to the greater hollowing of the supra- and infra-clavicular regions, for the most part accompanied with impairment or absence of mobility of the upper third of the chest. It is to be considered, moreover, that the peculiarities of form of chest about to be described as characterizing phthisis, are only manifest where incipient, however slight, deposition of tubercle takes place before the human frame has attained its full growth and maturity. But such general and vague information as is gathered from the patient's outward appearance, can serve only to direct the practitioner's attention to the danger of impending disease, and therefore suggest a more precise diagnostic investigation. Every sound pathologist must be well aware that phthisis attacks not alone those of "a consumptive habit," but even broad-shouldered and seemingly robust individuals.

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[1] Out of 122 cases of recent and well-marked tubercular disease of the lungs, I observed 14 preceding the 20th, 74 intervening between the 20th and 40th, 26 between the 40th and 60th, 7 between the 60th and 80th, and 1 subsequent to the 80th year.

On closely examining the thorax of phthisical persons, one cannot indeed fail to discover a certain degree of narrowness, which is, however, to be regarded not as a primitive defect, but as a symptom—a consequence—of disease within the texture of the lung. The narrowness is perceptible, in relation either to the broad diameter, as in the general phthisical habit, or else to the long diameter, as in the ordinary scrofulous build. In the former case the chest appears elongated, without being so in reality,—the false ribs failing to maintain their natural degree of expansion, and thus depriving the cone-shaped thorax of its usual breadth of base. By the ribs thus sinking down, the chest acquires more of a cylindrical character, appearing contracted, both at its apex and at its base, whilst the inferior intercostal spaces widen, and the epigastric region assumes a lengthened, groove-like shape. The abdominal cavity is at the same time diminished, and the organs, situate above, forced downwards. In the second case, the shoulders are indeed broad, giving to the thorax, superiorly, an appearance of ample space; and the inferior ribs are, at the same time, elevated, affording full room to the base of the lung. Still the length of the thorax by no means corresponds to the undue expansion of the abdomen,—the latter being inordinately wide and long,—the former short, and not broad in proportion. These deductions are the result of comparative measurements of the length of the sternum, and of the space intervening between the ensiform cartilage and the symphysis pubis, as also of the length of a line descending, on the outside of the nipple, from the clavicle to the superior margin of the liver, (which may be readily detected by repeated percussion,) and of another line drawn from the superior margin of the liver to the anterior, superior spine of the ilium. Individuals, in whom the aforesaid relations obtain, have, for the most part, been afflicted with strumous disease of the abdomen, before the period of puberty, and have generally a fatty liver and considerable tuberculous deposits within the bronchial glands. As before stated, however, tubercles, and also phthisis of the lungs occur, independently of those alterations of capacity in the thorax. Such examples are, however, rare, and, as will not seem surprising, after what has been advanced, more common in middle and in advanced age than in youth.

The influence of different climates upon the development of pulmonary tubercles, was formerly much overrated. An opinion prevailed almost universally that phthisis was far less common in southern than in northern latitudes, and particular localities, as the coasts of the Mediterranean sea, the island of Madeira, &c. were specially recommended as suitable for consumptive patients. We shall presently see how far this accords with the statistics of mortality from phthisis. Sir James Clark (l. c.) and Dujat (Gaz. Méd. 1838, No. 5,) have collected many interesting facts. From their statement it results, that in the East and West Indies, at Rio de Janeiro, and in Madeira, the disease is precisely as frequent as in Europe. From Annesley's observation (see Diseases of India, London, 1828), that amongst the British troops in the West Indies a greater number of black soldiers die of phthisis than white, we are not to conclude that the Ethiopian race is more prone to tubercular disease of the lungs than the Caucasian—since many of the Europeans, who would otherwise probably have thus died, fall victims to intercurrent tropical disease, to which the black population, is less obnoxious. This explanation would seem to apply equally to the fact, that amongst the white people of Rio and of the West Indies, the Europeans are more exempt from the disease than the creoles; and again, that (see Clark, from Marshall) at Ceylon, natives become attacked with greater relative frequency than Europeans. These statements, then, offer as little in favour of the climate of the tropics generally, as of specific changes of climate in particular. In what degree such changes would seem to promote the development of phthisis, may be gathered from the reports of the army surgeons of Great Britain, (collected by Major Tulloch). These reports, founded upon the average of observations extending over a period of 7 years, present the startling result, that out of an equal number of proportionately robust individuals of the same age and sex, living upon nearly the same diet, subject to the same general regulations, and exposed to the same amount of fatigue, the mortality from tubercular phthisis is greatest in the West Indies, and least in Canada and Nova Scotia; and that, at the Mediterranean stations, the proportion is almost exactly the same as in Great Britain; being at Gibraltar as 1000 to 8·2,—to 6·7 at Malta,—to 5·3 on the Ionian Islands,—and to 6·6 in the United Kingdom. The same report shows the mortality from phthisis, amongst the Maltese inhabitants, to be almost identical with what it is in Sweden, namely—at Malta as 1000 to $5\frac{1}{8}$,—in Sweden as 1000 to $5\frac{3}{5}$. Further testimony might be added, showing the disposition to tubercular disease to be nearly equal in all climates, did not the above remarks already border too closely upon digression. The chief practical inference deducible from these statistics is, that, under auspicious circumstances, a judicious change of residence,—for example, from a volcanic to an alluvial soil,—from a calcareous to a sandy district, and the like,—may prove serviceable, in incipient phthisis,—and again, that sea voyages (as shown by Dujat, and previously by James Johnson—On Change of Air, &c.) are often productive of the best effects.

Having seen, from the foregoing, that phthisis pulmonalis spares no age or bodily conformation, and that it is, moreover, almost uniformly frequent and fatal throughout the globe,—we shall now proceed to inquire into the precise nature of tuberculous predisposition, and into the causes of its morbific development. Its contagious and hereditary character, and its relation to mental influences, are points, which neither anatomical facts nor the statistics of its mortality, can clear up.

Where a real predisposition prevails, tubercles become developed in the lungs, under every variety of circumstances. Sometimes they form very rapidly, almost suddenly, and prove fatal, without previous destruction of the pulmonary texture, solely through disturbance to the respiration, as in tubercular hepatization (see Chronic Pneumonia), and in acute tubercle. At other times, the rise and progress of the affection are more gradual, and essentially chronic. The latter case is by far the most frequent, and its multiform relations merit the utmost attention. In some instances the tubercular development advances—obviously under the *exclusive* influence of existing predisposition—

so slowly, and its ulterior stages follow so insensibly, that the mischief escapes ordinary observation, until a very late period. (For latent cases see Louis' works, and also Andral's Clin. Méd., vol. iv, p. 145.) For the most part, the disease is traceable to a catarrh, which, after a first attack, leaves perhaps but a slight cough behind, but, on frequent repetition, gradually and irretrievably lapses into confirmed phthisis. The disease, almost equally often, sets in with hæmoptysis,—which recurs, at longer or shorter intervals, during its course. Generally speaking, chronic phthisis is wont to be interrupted by tolerably frequent breaks of seeming recovery, so that a limited number of tubercles may, under favorable circumstances, remain dormant for months, and even for an entire year, until an attack of catarrh or bronchitis, a slight pleurisy, or a peripneumony operates upon the already diseased parts, so as to revive the quiescent diathesis, exciting it to increased productiveness, and thus hurrying on the disease uninterruptedly to its fatal issue.

In chronic phthisis, death may take place gradually, in consequence of exhaustion and extinction of the respiratory function; or else suddenly,—at any stage,—from various causes. The fatal result ensues in many instances, from acute tubercular disease supervening upon,—and interfering with the course of,—chronic. At other times, inflammation in the texture of the lung unexpectedly closes the scene, either by throwing out a product of a tubercular type into previously healthy portions of the lung (tubercular hepatization), or else by encircling, individually, the numerous clusters of tubercle present, with a more or less thick border of red and gray hepatization. Occasionally life is shortened by pleurisy determining a mixed fibrinous and tuberculous exudation, which, by compressing the lung, mostly indeed prevents any further deposition of tubercle, but of itself proves mortal. Such pleuritic seizures may be the immediate consequence of liquefied tubercle perforating the pleura, and giving rise to pneumothorax. In rare instances, sudden death follows the rupture of a large branch of the pulmonary artery, that skirts or traverses a softened mass of tubercle,—profuse hemorrhage being the result. The same may be the effect of hæmoptoic infiltration of the pulmonary texture. Finally, the simultaneous deposition of tubercle in other organs,—the larynx, the intestines, the membranes of the brain, —oftimes carries off the patient ere the pulmonary disease has reached its acme.

*Acute tuberculosis of the lungs* demands a special consideration, as well on account of the peculiarities of its symptoms and course, as of the changes it presents on dissection. It seems to originate wherever, through existing disposition, the mass of fluids have become so saturated as, on a slight occasion, to throw out, amid tumultuous action of the arteries, an inordinate quantity of tuberculous matter. The most frequent occasion is a catarrhal affection; indeed, the disease seldom occurs without being thus preceded. Acute tuberculosis arises either in persons previously free from tubercle, or else in those in whom manifest traces of long extinct tubercular affection are discoverable, in some organ or other; or, finally, it is grafted upon phthisis, already present in a chronic form. Accordingly two forms may be discriminated, a primary and a secondary. The first assails youthful persons, between the ages of 18 and 25, more especially of the male sex; the latter, those in the prime of manhood, or even in advanced age. In very marked cases, one or both lungs are found, from the apex to the base, uniformly loaded with an extraordinary number of tubercles. These are always isolated granules, as big as pins' heads or millet seeds (miliary tubercles), mostly yellowish and soft, but occasionally grayish, and more solid. The colour and consistency depend upon the degree of irritation produced in the surrounding textures by the rapid deposition of tubercle. The yellow and soft ones are found in the centre of a group of red or gray hepatized pulmonary cells; while the gray tubercles are imbedded in a texture saturated with bloody serum. When the disease is somewhat slower in its course, the tubercles are less uniformly miliary, being in a great measure united into little groups, and more densely crowded at the apex, and in the superior lobe generally, than towards the base of the lung. Acute tubercular phthisis proves invariably and speedily fatal, very often during the third week. In two instances only of the primary form have I observed it protracted, once to the fifth, and in the other to the sixth week. In the latter case, it passed into a chronic state, and cavities formed. The vital symptoms are very peculiar, bearing so close a resemblance to those of typhus fever as to lead to mistakes. The diagnosis can only be determined by the stethoscopic signs, and sometimes by the continuance or frequent recurrence of hæmoptysis. On cadaveric inspection, one or both lungs are found greatly tumefied; they do not collapse when the thorax is opened, are very dark coloured, preternaturally soft, throughout, and gorged with blood and serum. The numerous and equably distributed tubercles have everywhere the same character, a sure proof of their simultaneous origin. They are for the most part surrounded, for a line's breadth, by inflamed, if not hepatized, parenchyma; the inflammation seldom, however, extends to any distance; thus, notwithstanding the great multitude of the tubercles, pulmonary cells, still permeable, and comparatively healthy, are everywhere cognizable. The bronchial canals are of a deep red, often passing into violet. Adhesions betwixt the pulmonary and costal pleuræ are never met with, unless of earlier date. Miliary tubercles are sometimes seen upon the pleura and other serous membranes; in rare instances, beneath the arachnoid. All the solids and fluids of the body suffer a change, inasmuch as a rapid exosmosis of serum is determined, and liquid effusion, tinged with blood, accumulated in all the serous cavities.

Where the disease has run a very rapid course, tubercles are not encountered in any organs besides the lungs. More frequently however they occur elsewhere; thus they are thrown out very numerously into the texture of the spleen, a few are seen upon the surface of the liver, others again in the cortical substance of the kidneys, and I have very often found the supra-renal glands so replete with recent tubercle, as to have increased to thrice their ordinary thickness. The mucous membrane of the intestines is but seldom implicated; when it is, individual cysts of Peyer's glands become the receptacles for the morbid matter. The greatly altered character of the blood, in this disease, is rendered obvious by its fluidity in the blood-vessels and the heart, by the imbibition, not only of the coats of blood-vessels, but also of the parts adjacent to the larger veins; finally by the softening of many organs, especially the heart and mucous membrane of the stomach. For the same reasons, the corpses of consumptive subjects are liable to unusually rapid decomposition. In two instances I found, in both ventricles of the heart, globular coagula containing pus-like fluid.

*The miliary deposit* of acute phthisis has, by some pathologists, been mistaken for the product of intense bronchitis, implicating the pulmonary cells. Cruveilhier injected mercury into the bronchi of dogs, and found, on killing them, the parenchyma of the lungs filled with a multitude of little granules, consisting of a tolerably solid puriform mass, with a central nucleus of mercury. He imagined that he had thus created artificial tubercles. The experiment is cited by all those who would assign an inflammatory origin to tubercles generally, or at least who, denying to the acute miliary form the generic characters of tubercle, would class them as products of inflammatory exudation, Dr. Addison, for example, (Guy's Hospital Reports, April, 1837). Exact microscopic research has however, quite confuted, this doctrine. Gluge (l. c. p. 59), who, with Magendie, repeated these experiments, as Andral and Lombard had previously done, has distinctly proved, by the aid of the microscope, that the substance surrounding the quicksilver consists solely of the so-called composite inflammation-globules and pus-corpuscles, and by no means of tubercle-cells or granules. On the other hand, the results of cadaveric inspection, as before detailed, are nowise compatible with the idea of inflammation of the lungs, but point, on the contrary, to a diseased state of the fluids generally. Hence bronchitis can only be admitted to be the contingent cause, and diffuse hepatization its consequence,—never the essential characteristic of acute tubercular phthisis.

A description of the organic changes, produced by true chronic phthisis, will still more distinctly show the disease to be one of a general nature, deeply rooted in, and generally pervading the constitution.[1] Where, as in the majority of cases, the development of tubercle is gradual, both lungs are assailed almost at the same time, though scarcely ever to the same amount; one of the lungs becomes sooner charged with tubercles, and, accordingly, falls an earlier prey to the destructive process. Louis observed, in 123 cases, predominant disease of the left lung;[1] but, in the cases of phthisis that occurred at the Leipsic hospital in 1839, I found the right lung the more frequently affected; Mohr, again, (l. c. p. 86,) asserts, that neither side is *absolutely* more liable than the other. Tubercular disease almost invariably commences at the apex of the lung, from whence it gradually descends, so as to present a decreasing scale of development from the summit downwards. This relation is very constant, as regards the upper and lower lobes, but does not altogether apply to the middle lobe. Thus, while one or more large cavities are met with at the apex, there occur, inferiorly, only smaller ones,—and, towards the base of the upper lobe, dense accumulations of tubercles, which have but partially undergone incipient softening. The relations of the lower lobe are the same, though on a reduced scale. At its upper part, there are small vomicæ; lower down tolerably crowded, but, about the base, merely scattered groups of tubercles, or, possibly, none at all. In two solitary cases out of 123, Louis found numerous tubercles in the act of softening, exclusively in the inferior lobe.

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[1] At a time when, in France at least, it was the fashion to derive almost every disease from irritation and inflammation, Louis confuted, by numerical evidence, the inflammatory origin of phthisis. See his 'Researches on Phthisis,' translated into English by W. H. Walshe, M.D., &c.

Pulmonary tubercles assume manifold aspects; we shall, however, distinguish but two varieties, in reference to the character of the individual granules, and three in reference to the manner in which they are grouped and congregated.

The two former are the *gray* and the *yellow* tubercles. The gray are by far the most common; they have the colour and transparency of coarse scalded groats; they are far more firm, however, and tolerably renitent on pressure. Their size is uniformly intermediate between that of a grain of mustard and a grain of millet seed. This has been supposed, especially by Laennec, to be the primary form of tubercle, generally, and that which before softening merges into the next. But an unbiassed examination of tuberculous lungs shows it distinctly to be an independent variety, which, in numerous instances, passes through all the ulterior stages. Previously to its softening, this tubercle loses, indeed, some of its transparency, and may then occasionally assume more of a yellowish hue, but this is, for the most part, owing to purulent secretion derived from the surrounding textures, in which inflammatory irritation engenders, or at any rate accompanies the further changes of the morbid deposit.

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[1] With respect to the predominant liability of the left lung, Louis states that, of the cases where only one lung contained tubercles, the left lung was five times,—the right but twice diseased. But it should be remembered that acute phthisis, which he does not consider separately, observes different relations.

*Yellow* tubercles are thoroughly opaque, of slight consistency, and of a dull straw colour. They are generally isolated, and most strongly characterized in acute phthisis. They vary in size, but for the most part exceed that of the preceding kind, and may attain the bigness of a hempseed. All the other forms of tubercle, mentioned by different authors, may be brought under these two heads; for either they are mere transition-forms, or simply gray or yellow tubercles, variously modified, during the advance, or the retrogression of phthisis. This applies specially to the so-called encysted tubercles.

Where tubercles are spoken of, much larger than those above described, a congeries must be understood. For I, myself, at least, never met, in the lungs, with any single ones bigger than grains of hemp seed. The two forms described rarely coexist in the same lung, and where they do, their deposition is evidently referrible to perfectly distinct epochs.

*The gray semi-transparent granulations* of the French pathologists are deserving of separate notice. They are to be regarded either as belonging to the *gray* variety of tubercle, or else as secondary deposits, not necessarily invested with the character of tubercle. They originate almost exclusively at an advanced period of phthisis, and in the vicinity of former tubercles,—being, in all probability, a simple product of the inflammatory irritation occasioned by the latter, in the texture of the lung. Where that irritation is great, the substance effused is presently converted into pus, to the speedy destruction of the texture itself. Where it is only slight, and the tubercular diathesis has become latent or extinct, the effused product becomes organized, and shrivels, by degrees, into the cyst wherein the tubercle is isolated. Where, again, a limited grade of irritation persists under a still prevailing tendency to tubercular action, the exudation possesses the elements of eventual transformation into genuine gray or yellow tubercle. It is only under the last-named circumstances, that the gray, semitransparent or gelatinous exudations can be regarded as a first step towards the formation of tubercle.

With reference to the mode in which tubercles are distributed through the lungs, three distinct relations obtain. First, they are found single, isolated, and more or less uniformly disseminated (*miliary tubercles*). Secondly, they are found in scattered groups, assuming various forms, the tubercles being now loosely collected together, now closely connected, either in a regular mulberry shape, or in clusters of indefinite form (*aggregate tubercles*). Thirdly, and lastly, they are found so densely crowded, throughout a portion, if not the whole, of a lobe, as to constitute, seemingly, but one coherent mass (*tuberculous infiltration*). Their mode of distribution is naturally influenced by their mode of development. Where they form rapidly, they are the more equally dispersed; where slowly, they become in the same measure subject to the law which causes them to accumulate in the summit of the lung; and, from thence downwards, gradually to decrease in compactness, with a proportionate tendency to run into groups. The infiltrated form, alone, generally appears to set in all at once, in an acute way, and with the character of hepatization. The aspect of the individual tubercles is also modified by the manner of their distribution; the gray variety, in particular, becoming whiter, and losing its transparency, when densely congregated. For the most part, tubercles are limited to a single mode of distribution—in the lungs of an individual; still cases occur in which all the three forms, before described, are met with in the same lung, each form being, however, obviously due to a distinct period of deposition.

Concerning the structure and elementary composition of tubercles, great diversity of opinion has always prevailed. In general terms they may be said to consist of a perfectly inorganizable mass, the analyses of which, though varying somewhat, according to different standards of organic chemistry, present on the whole no very great discrepancies.[1] The chief organic constituents are fibrin, casein, fat, and a very small proportion of albumen; the inorganic components are, chloride of sodium and phosphate of soda, phosphate and carbonate of lime, some little oxide of iron, &c. The peculiar manner in which some of the organic materials have been found combined in tubercles, has given rise to the notion that they contain phymantine and pyine. But this is as yet by no means certain, Preuss and Güterbock being still at variance concerning the relations of pyine, of albumen, and of casein. The changes which tubercles subsequently undergo, modify their chemical composition.

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[1] The different analyses are to be found in Vogel (über den Eiter. u. s. w., 1838, pp. 114–116; and in the addenda thereto, p. 229.)

The results of the microscopic examination of tubercles vary greatly with their different stages of development. Freshly deposited tubercles consist of a mass closely resembling recent inflammatory exudation. We have a grayish, dull, somewhat homogeneous, and amorphous substance, which presents a granulated appearance, and in which a multitude of granules can generally be recognized. This mass differs from the product of inflammation, in being tougher and less easily separable into parts, insoluble in, and scarcely even rendered transparent by acetic acid. More mature tubercles, however, especially of the yellow variety, which have begun to assume a curd-like character, and are more easily broken up, are less amorphous, and more distinctly granulated, whilst they at the same time present several spherical, and occasionally oval, bean-shaped, or serrated cells, some smaller, others larger than pus-cells, and somewhat flattened. These cells are filled with an indefinite number of, for the most part, equal sized granules, about $\frac{1}{2000}$ of a line in diameter; they still remain opaque, however, and compactly imbedded in the above amorphous mass. Neither acetic acid nor solutions of neutral salts produce any perceptible change, whereas concentrated mineral acids and solution of potash soon destroy them. In tubercle fully engaged in the process of softening, these cells are very conspicuous; readily become isolated, are larger, and in general display an irregular bean-like, serrated, or angular shape, having at the same time become more transparent, so that their granules are more visible. After the complete liquefaction of tubercles, only a few cells are discernible, but, in the midst of a quantity of adventitious substances, are seen their almost entirely denuded contents,—those granules, namely, the diameter of which does not exceed 0·0008 of a line. Some of them are irregularly crowded together, others isolated, and all suspended in a limpid, or slightly turbid, grayish fluid. The shrivelled, mealy, or moist chalk-like remains of tubercle, consist almost entirely of dense aggregations of the granules, mingled almost invariably with cholesterine crystals, and occasionally with remnants of the cellular tissue of the lung. Together with these peculiar elements of tubercle are found, according to circumstances, pus-corpuscles, granulated cells, ciliary epithelium, cellular-tissue fibrils, and even caudate cells.

It frequently happens, as Laennec fully demonstrated, that the interior of a tubercle is softer than its outer surface, and exhibits in its centre a dark-coloured nucleus, formerly believed to be the point where the after process of softening commences. There is, however, as the sequel will show, little reason to doubt its being in reality a mere residue of the original contents, morbid or natural, of the air-cells. The tuberculous matter being first deposited on the walls of the vesicles, gradually presses towards the middle, (and that in very short time,) the epithelium-cells, or whatever else the vesicle may contain, and then incloses them as a kind of soft and sometimes dark-coloured nucleus.[1]

The numerous researches hitherto made have not sufficed to determine the exact seat of tubercles within the lungs. The point is indeed beset with great difficulties, more especially when it is remembered, that certain forms of deposition are attended with organic change in the adjunct textures. Accordingly it is almost impossible to establish the precise seat of the morbid product in acute phthisis, or in tubercular infiltration. If, however, it be borne in mind, that tubercle is first thrown out as an exudation, out of which cells are only subsequently developed, and that the exudative process occurs most readily on membranous surfaces, and within free spaces, we must allow the pulmonary vesicles to be a very fit locality for accumulation, of those morbid cells, visible to the naked eye as minute tubercles.

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[1] A fact observed by John Home, and recorded in the Edinb. Med. and Surg. Journ. (Jan. 1838,) is highly interesting, in relation to this point. In a stone-mason, who died of phthisis, was found, at the centre of many of the pulmonary tubercles, an earthy nucleus, which turned out to be of precisely the same character as the stone of the Craigleith quarry, where the man had been employed.

*In acute tubercular disease,* however, where they are rapidly developed, and always mingled with pus-corpuscles, the morbid cells cannot fail to occupy both the free area of the vesicles and their walls. Each vesicle thus becomes augmented in bulk, pressing, it would seem, upon the neighbouring vesicles, on which account the miliary tubercles, especially the yellow ones, connected as they are with more violent inflammatory action, are seen by the naked eye to equal, if not to exceed, the presumed magnitude of the air-vesicles themselves. From this may be inferred the difficulty of ascertaining the true seat and limitation of these isolated tubercles. Where, however, the deposition is *preceded* by degeneration and condensation of the pulmonary texture, the difficulty amounts to impossibility. This applies equally to tuberculous infiltration, where all the areas fill almost instantaneously with the morbid sustance, the intervening textures being so compressed thereby, as to be no longer distinguishable. Here the cavities of the pulmonary vesicles obviously become the first receptacles for the morbid product.

It is only in the instance of aggregated tubercles of slow growth, that careful dissection affords rather more positive results. We are chiefly indebted to the exactitude of Carswell's researches for being able to pronounce with certainty that the pulmonary vesicles are the original seat of this form, a circumstance that does not exclude the possibility of deposition within the parenchymatous texture, at a more advanced period of the disease. Tubercles take the mould, so far as outward form is concerned, of the textures in which they are imbedded; hence, in the lungs, they assume the spherical shape of the pulmonary cells. They are, however, not regularly spherical, but rather acuminated, in proportion as they project more or less into the minute bronchial twigs. When deposited in large groups, and occupying numerous cells of a lobule, they resemble, in the aggregate, a little sprout of cauliflower, as is well figured in Carswell's plates. (Fasc. Tubercle, pl. i, figs. 1, 2, 3.) The pedicle of such a congeries of tubercle, occasionally extends far into the implicated bronchial twig. This disposition of the morbid substance is often apparent in sections when successfully made, but still more so in fragments of sodden or carefully macerated lung. The same thing may be seen, at times, on tearing asunder tuberculous lungs, when just beginning to soften, through incipient inflammation. Comparative pathological anatomy furnishes corroborative evidence. In the lungs of ruminating animals, especially of neat cattle, the tubercular masses are often seen extensively plugging up the bronchial tubes; these, dilated thereby, produce, chiefly it would seem by compression of the surrounding substance of the lung, the remote results of destructive inflammatory irritation. Carswell supports his view by a reference to the analogous behaviour of tuberculous deposits, in other hollow organs, invested with a delicate mucous membrane.

If, in accordance with the foregoing, tubercle be regarded as a morbid secretion incapable of organization, and deposited for the most part within the pulmonary vesicles, there will be little difficulty in comprehending the changes that follow. Either the deposit suffers no change beyond the absorption of its aqueous parts, and remains latent as solid tubercle for an indefinite period—which is, however, only the case where but a few scattered groups have formed, and that slowly—or else a great multitude rapidly congregate in compact masses, and there ensues more or less irritation of the surrounding pulmonary textures, followed by softening of the tubercles. It was once the general belief that this change proceeded from the middle; because the soft black central point was almost always observed to be present. Accordingly, tubercles were held to possess a certain vitality of their own (growth by intussusception), and also the inherent germs of decay. But it was eventually ascertained that, in the majority of cases, tubercles act as foreign bodies upon the surrounding textures, exciting in them inflammation followed by suppuration, which, reacting upon the tubercles, cause them to soften from their periphery inwards, and ultimately to become removed by expectoration. Sometimes, indeed, a spontaneous breaking up of the cells of a tubercle causes the central portion to soften first. Even then, there must needs supervene irritation of the surface, and consequent effusion,—the liquid portions whereof are imbibed by the tubercle-cells, which thus become distended, and burst asunder. But wherever the tubercular diathesis prevails, the effusion from the surrounding parts is not purely inflammatory; hence not only is recent tubercle deposited in the room of what has been withdrawn, but the disease gradually spreads to other and fresh portions of the lung, so as, in due time, to involve an entire lobe or more. As individual tubercles may be found, either surrounded with healthy texture, or else liquefied by suppuration of the pulmonary cells,—in like manner, a lower grade of inflammatory irritation may create thickening and induration of air-vesicles loaded with tubercle. This is brought about as follows: plastic exudation, sometimes gelatiniform, sometimes of a more firm consistence, is deposited for one or more lines' breadth upon the circumference of the single tubercles, or round whole groups of them, causing obliteration, hardening, and shrivelling of the involved air- cells. The tubercles thus acquire the appearance of being inclosed within a proper cyst; under such circumstances they are, in some sort, insulated from the healthy textures, and may long remain dormant, until quickened by some fresh source of irritation,—catarrh and the like. Where an entire cluster is thus encysted and compressed, the included textures disappear after a while, and the whole assumes the aspect of a single large tubercle.

In some instances the inflammatory action, wherewith the softening of tubercle is mostly associated, is not confined to the vesicles immediately surrounding the morbid deposit. In other words, vesicular pneumonia is no longer restricted to a number of distinct patches, separated by healthy lung, but overspreads an entire lobule, or even a lobe. As a general rule, death speedily ensues where the tubercular destruction is already extensive, and the general strength greatly reduced,—indeed, before the first stage has passed over. Where the tubercular mischief is more limited, the fatal issue is preceded by red or gray hepatization of the involved parenchyma. Tubercles already present in inflamed textures, soften very rapidly under these circumstances,—forming pus-like accumulations as big as hemp-seeds or peas, which, on incision, pour forth their contents, and exhibit a cavity, lined with a soft yellowish membrane. Sometimes the inflammatory product is deposited simultaneously with an excess of a tuberculous matter, and so intimately united with it, as to form but one whole (see Pneumonia). A lobe thus affected is of a grayish or yellowish white hue, thoroughly impermeable, and of a firm, brittle nature. Here, only such tubercles as existed previously to the textures becoming inflamed, are found softened.

In proportion as the tubercular degeneration spreads through the lung, corresponding adhesions almost invariably form between the pulmonary and costal pleuræ, commencing at the apex of the lung, and progressively descending, so as often to implicate the whole of the pleural surfaces. Similar adhesions connect together the different lobes, and ultimately unite the base of the inferior lobe with the serous covering of the diaphragm. These in time become so firm as to render their separation, with a view to the removal of the lung, a very difficult matter. So constant is their occurrence in phthisis, at the apex of the lungs at least, that I have but thrice failed to find them, during a period of four years. The pleura here loses its serous character, and suffers various alterations, to be hereafter specified. As the softening proceeds, cavities or vomicæ, of various dimensions, become developed, and an entire lobe, nay, the two thirds of a lung is occasionally converted into a single excavation. For the most part, however, the destructive process is arrested at the confines of a lobe, without breaking through the interlobular adhesions. The development of the cavity is due, in the first instance, to the softening and liquefaction of the walls of an obstructed air-vesicle, and of those immediately adjoining, through purulent secretion. The little excavation is, at the outset, invested with a delicate and yellowish adventitious membrane, of the consistence of pus; behind which fresh purulent and tuberculous matter is being constantly secreted, owing to progressive inflammatory irritation. Thus, no sooner is the original false membrane liquefied, than a new one forms in its stead, and the cavity enlarges proportionally. During the amplification of the vomica, the adjacent parenchyma of the lung undergoes sundry changes, the whole circumference of the cavity being found,—to the depth of a line or more in the direction of the healthy textures,—soft, moist, dense, non-crepitant, and of a dirty-gray aspect; in short, in a condition analogous to hepatization. As the tubercular softening and excavation mostly begin at several points at once, it frequently happens that very small intervening patches of the involved lobe continue sound, although the whole affected portion, generally the apex of the lung, remains quite impermeable and unfitted for respiration. This may depend on the compression exercised by the tubercular deposits, and by the hepatized texture which surrounds the cavities, upon the remainder of the pulmonary vesicles, or rather upon the minutest bronchial twigs; or else these twigs themselves become choked up with tubercular secretion deposited on their walls, or within their canals; or, again, vesicles, not directly within range of the tubercular action, fill with gelatine, which, gradually solidifying, assumes a reddish tint, and ultimately transforms the pulmonary cells into a firm, red-brown, smooth, or exceedingly fine granular substance. When the disease proceeds in a less rapid and less equably progressive manner, now and then pausing, and coming to a standstill, the membranaceous lining of the cavities assumes quite a different character,—indicative of a sanative effort, which now and then proves successful. These false membranes are tolerably consistent, frequently of almost semi-cartilaginous hardness,—now of lardaceous, now rather of a fibrous aspect,—their inner surface being at first rough, uneven, or eroded, but assuming, at a later period, a velvety look. Such investing membranes are capable of gradually becoming vascular and organized,—as we shall afterwards see, in a very peculiar manner. The *velvety* lining, in particular, consists almost exclusively of very delicate, newly framed blood-vessels, through whose medium, provided the tubercular diathesis be lulled for an interval, if not permanently,—the membrane is converted into a pus and mucus secreting structure. The said membrane is formed out of the hepatized crust that encircles the enlarging cavities, and which, instead of becoming softened and liquefied through purulent formation, merely becomes saturated with coagulable substances more akin to the organism. But, no sooner is the tubercular diathesis revived, and aggravated by a catarrhal or inflammatory attack, than the protecting false membrane liquefies, and purulent secretion, mingled with tubercle, again sets in. The cavity itself, which, under the above circumstances seemed to shrivel and contract, gains size; new vomicæ form, the lung becomes more and more deeply involved, and the mischief terminates only with death.

The cavities, while still small, are spherical; by and by, however, they acquire a very irregular shape, either because several adjoining ones unite, or else because the softening and liquefaction of the contiguous textures proceed more rapidly on the one side, than on another. For this latter reason, cavities are seen to bulge out on every side, or else to communicate, through narrow channels, with other cavities; and patches of lung are found undermined, in every direction, in the midst of a cavity. Occasionally such fragments of lung, of a most fetid character, are expectorated during life. In some few instances, again, the fragments being, by the joint effects of tubercular destruction and of infiltration, totally cut off from the circulation, slowly mortify and slough away. Here death very speedily ensues, the adjacent parts being too thoroughly disorganized to admit of repair through separation and expulsion.

Unless the disease be on the decline, several bronchial tubes invariably open into the large-sized cavities. This communication of the air-passages does not, however, exist in the first instance; because the incipient softening of tubercles, whether alone or in groups, being generally attended with inflammatory action, all the adjacent textures, as above stated, become filled, and consolidated with plastic materials, causing closure of the implicated bronchial tubes, and isolation of the vomicæ. It is to be noted, that the smaller, simply membranaceous, bronchial twigs, are, at the same time, mechanically compressed by the increasing mass of tubercle, and often obstructed with it. This accounts for the fact, that, generally speaking, the bronchial tubes are not found to open freely into the lesser tubercular cavities, unless where their compression and closure are prevented, either by greater width of the caliber, by some morbid alteration of their walls, or by the help of a cartilaginous framework. The destruction of the bronchial canals is contemporaneous with that of the other textures; wherefore they terminate abruptly at the brink of a cavity, and seem to blend with the walls. But even in the later stages of phthisis, various contingencies, as the presence of a clot, of tough mucus, of imperfectly liquefied tubercle, and the like, may give rise to temporary obstruction of the inosculating bronchia. In such instances, the sudden reopening of the outlet now and then occasions overloading of the air-passages, and death by suffocation. (See Andral, Clin. Méd. vol. iv, obs. 4.)

*The nerves* have been traced by Schroëder van der Kolk (l. c.) up to the walls of a cavity, where their minute fibrils become swollen, and the neurilemma reddened; beyond this point they blend with the lardaceous, softened, or hardened texture, and are lost to sight.

The relation of the blood-vessels, in tubercular disease of the lungs, is of paramount interest. Whenever a portion of lung becomes impermeable to air, and therefore disqualified for its natural office, its functional blood-vessels retreat, and are replaced by vessels from the aortal circulation. The respiratory circulation ceases, and the general circulation establishes itself, in a corresponding measure. This is effected by obliteration of the pulmonary, and by augmentation of the branches of the bronchial, arteries; the internal and external mammary, and the intercostal arteries, at the same time communicating, by new and often very extensive branches, with those portions of lung which are now, on every side, firmly adherent to the thoracic walls. In many instances, this state of things reveals itself during life, by the extraordinary development of the subcutaneous network of veins, in the clavicular region. Under the above circumstances, the intercostal arteries will frequently take up an injecting mass from the pulmonary artery, and the latter from the aorta, whilst the blood would appear to have been returned to the heart both by the intercostal and the pulmonary veins. But these anastomoses are by no means constant; the two circulations often—I should myself say, almost invariably—remain distinct. We are indebted, for an explicit detail of these interesting facts, to Schroëder van der Kolk (l. c. fasc. i, pp. 74-85), whose observations have since been confirmed by Sebastian (De origine Phthiseos Pulm.) Similar results have been deduced from a triennial series of observations made at the Leipsic hospital, as also from the injections and microscopic researches of G. N. Guillot (l'Expérience, 1838, No. 35.) The obliteration of the minuter twigs of the pulmonary artery speedily follows the deposition of tubercles; for when an injection is thrown into the pulmonary artery, it rarely happens that it can be propelled into the immediate vicinity of the crude tubercles; Guillot observed, in particular, that the walls of pulmonary cells, which have become the receptacle of tubercle, form a coriaceous layer, sometimes two fifths of an inch thick, wholly impenetrable to the pulmonary arteries, and presently supplied with new vessels. The more equably the pulmonary textures become disorganized, the more completely are the twigs of the pulmonary artery obstructed, up to the larger trunks. The more perfect the adhesions on the surface of the lung, the more readily is a junction with the arteries of the aortic circulation established. Closure of the pulmonary arteries takes place here, precisely as in the instance of the fœtal vessels, and of phlebitis, although without there being any ground for our assuming vascular inflammation.[1] The walls of these vessels thicken, or swell quite disproportionately,—the several tunics merge into a single, uniform, sero-lardaceous mass; and the caliber, which long continues narrow, though pervious,—becomes eventually filled with a thin, reddish, fibrinous plug, transforming the whole vessel into a solid cord. These cords either form a coarse network, covered with remnants of lung, and invested with a purulent crust, upon the walls of a cavity, or traverse the latter as bands or bridles. In rare instances, a cord of this description tears through attenuation, midway between its two ends, before definitive closure of the caliber. Then ensues more or less violent hemorrhage, which often proves suddenly fatal; the vomica, and the bronchial tubes corresponding, are found replete with coagulated blood, which probably fills, in a frothy state, the trachea and larynx.

The above explanation of the deportment of the blood-vessels will at once account for the hemorrhage so frequently attendant on tubercular disease. It is often considerable at the outset, and recurs, from time to time, during the progress of phthisis, although not to such an amount as immediately to threaten life. It is chiefly observed where tubercle is rapidly developed, in connexion with inflammatory action, or where the pulmonary texture is replete with densely-clustered tubercles. Carswell is, upon the whole, right in stating that the blood is poured out because the pulmonary veins, being compressed by the multitude of tubercles, are not in a condition to take up the whole of the arterial supply.

Although the spread of a cavity is generally confined to the internal texture of a lung, it occasionally perforates the pleura, engendering pneumothorax, or, in very rare instances, pulmonary fistula, which extends to the external integument. The intimate adhesions betwixt the tubercular portion of lung and the costal pleura, for the most part, obviate the occurrence of pneumothorax; yet examples are not wanting in which those adhesions being incomplete, a small superficial cavity leads to sloughing and destruction of the pleural covering. The resulting perforations are never large, exhibiting mostly a narrow fissure or oval aperture, three or four lines long, in the midst of a very soft, dirty, yellowish or grayish texture, which gives way and tears under the slightest pressure. They are generally situate in the neighbourhood of the third or fourth rib, and somewhat laterally,—in the direction of the axilla. In 8 instances of pneumothorax, Louis found the left side 7 times affected. I have myself, within a period of four years, met with pneumothorax 9 times on the left, and 7 times on the right side. Reynaud collected 50 cases; 33 of which were on the left, and 17 on the right side. (See also Puchelt on Double pneumothorax, 'Heidelb. Annalen.' vol. vii, fasc. 4.) Very shortly after perforation has occurred, violent pleurisy sets in, and life is promptly extinct, although occasionally prolonged for a week and upwards. Where the fatal issue is farther delayed, the perforation must needs be surrounded by adhesions, which, by confining the mischief within narrow bounds, render the consequences comparatively harmless. On cadaveric inspection are found the different marks of acute pleurisy, namely, granular investment of the whole serous surface, with a grayish and very soft mass,—purulent fluid, and dirty, yellow, purulent false membranes; all this is coupled with a fetid accumulation of gas, which, upon opening the thorax, often escapes with a hissing noise. The lung is always compressed to the utmost, unless when kept permanently distended by adhesions. Near the perforation the effort of nature to repair the injury, by coating the part with coagulable materials, is very evident.

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[1] Nor is the case to be confounded with true inflammation of individual branches of the pulmonary artery, not rarely the result of general inflammation of the pulmonary textures, supervening upon phthisis.

The cavities above described belong for the most part to chronic phthisis; other conditions prevail where the softening takes place within lungs densely infiltrated with tubercle. Here the general mass liquefies at several points all at once; irregular clefts form in all directions, together with cavities neither sharply circumscribed, nor invested with the membranaceous tuberculo-purulent deposit before described. The softening makes very rapid progress,—the entire mass crumbling and liquefying, and exhaling, even during life, an intolerable fœtor. At this conjuncture death almost always closely follows.

Having duly considered tubercle in its destructive character, we shall next inquire into the circumstances under which it is rendered inert, and its ravages repaired by the healing process, even at an advanced period of the disease. The retrogradation of phthisis rarely takes place between the eighteenth and thirty-sixth years. Consumptive persons, however, who have attained maturer age, and ultimately succumb from disease of another kind, or from mere senile debility, offer more or less proof either of entire recovery from tubercular phthisis, or at any rate of diverse efforts of nature to bring that about. Even at such a period, the disease runs its resistless course whenever the number of tubercles is excessive, or the causes productive of renewed deposit act with but slight interruptions. In old age, again, the tubercular diathesis is sometimes revived with new energy after a long interval of repose,—rapid consumption being the consequence. An example of this kind was observed at the Leipsic hospital, in a female aged eighty-nine.

It is still matter of doubt, whether tubercles can be removed by absorption, while in a crude state. Andral and Carswell believe the thing possible, and their view is certainly favoured by the circumstance that, while a tubercular cavity is for the most part numerously surrounded with crude tubercles, a portion of lung, containing the obvious remains of a cicatrized cavern, sometimes presents hardly a vestige of them in their primitive condition. There is here more likelihood of their having been absorbed than expectorated.

The healing process is especially conspicuous, where changes, tending, under less propitious circumstances, to softening and excavation, have already influenced both the tubercles and their vicinity. Two conditions are quite indispensable to perfect cicatrization, namely, first, that there be no great amount of tubercles present; and, secondly, that the incipient softening be not, as it more usually is, attended with the continued deposition of tubercle.

The cure of pulmonary tubercle, whether in the crude or the softened state, is more particularly due to shrivelling and calcareous transformation. These changes are, however, as before stated, mostly preceded by inflammatory effusion, of a simply plastic nature, and free from all tubercular taint, into the adjacent pulmonary cells. On the subsidence of inflammation, the involved textures shrivel in a degree corresponding with the obliteration caused by the same process in the minute bronchial twigs. Ultimately the tubercular mass is simply surrounded with a thin, isolating crust, and no sooner is it within range of newly-formed blood-vessels, than the absorption of the fluid parts of the tubercle gradually ensues, leaving but a dryish, pap-like residue behind. The organic elements of tubercle would moreover appear to be at the same time taken up by the system, and phosphate and carbonate of lime to be progressively deposited in their stead. Now and then the deposition, more especially in youngish individuals, takes place somewhat hastily; so that the outside of the tubercle is alone changed into a calcareous crust, while the central part retains its softness. Sometimes again the calcareous matter is thrown out at certain intervals, producing a series of distinct, hard, superimposed strata. Thus we see that a tubercular mass is not always thoroughly converted into a uniform, hard concrement. Most commonly, it would seem that when the process advances slowly, the tubercles put on the semblance of moist chalk. Thus the original volume of the entire mass continues to diminish—so much so at times, that a considerable portion of lung, as may be inferred from the size of the bronchial tubes leading thither, becomes reduced, by obliteration and shrivelling, to a hard shell, holding in its centre a chalky tubercle no bigger than a pea. The healing process just described is by no means rare, its traces being often found in the lungs of very aged persons who have died of various maladies.[1] It is likewise met with in younger individuals, though much less frequently.

Tubercular cavities heal precisely in the same manner, whether debarred from the air-passages or connected with several larger bronchial canals. In the former case we have exactly the same capsule resulting from inflammatory induration of the surrounding texture, wherein the inclosed tubercular mass is converted, first into a chalky pap, and eventually into a hard calcareous concrement. The majority of these concrements originate in the lesser and still closed cavities. They are generally irregularly shaped, and of a rough granular surface; sometimes quite hard, at other times friable;—and often contain mealy or pap-like nuclei. They occur most frequently at the summit of the upper and of the lower lobes, where they are firmly impacted within a shrivelled and indurated texture. In rare instances the residue of tubercular matter, not removed by expectoration, but left within the larger cicatrized cavities, is, by degrees, converted into earthy granules. These are loosely held within the scar-contracted cavity, where they mingle with tubercular and muco-purulent fluids, and may become ejected during a violent fit of cough, provided the implicated bronchial tubes still remain open.[1]

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[1] Rogée, whose researches respecting the residue of tubercular disease are valuable, met with these concrements in 51 out of 100 aged subjects (see Arch. Gén. Juin 1839.) See, also, Andral (Clin. Méd. vol. iv, 4$^{me.}$ édit. p. 137).

Cavities cicatrize in a variety of ways. They may disappear altogether, or contract only to a limited extent. In the former case they fill with a cellulo-fibrous substance. We have before seen that when fresh deposition of tubercle,—in other words, the destructive process,—is arrested, the parietes of a cavity may become invested with a comparatively hard, organized membrane. This seems to be the first step towards cicatrization. For, while the pre-existent soft, tuberculo-purulent false membrane, as an unorganizable deposit, is being continually destroyed, and replaced by new and similar ones under progressive enlargement of the cavity, the membrane in question is assimilated to the organism, and neither liquefied nor ejected, but gradually thickened. It is difficult to decide whether this thickening proceeds from behind, or from plastic effusion upon the free surface of the membrane; the former is, however, the more probable assumption, because the free surface, although vascular, generally presents the velvety aspect peculiar to mucous membranes. True granulations of the well-known form and magnitude are not distinctly cognizable upon the surface. By dint of thickening, the membrane grows, as it were, towards the centre; thus the cavity contracts more and more, until at length it fills with a cellulo-fibrous substance resembling brawn.[1] The gradual effacement of the cavity is further promoted by the condition of the neighbouring parts. When, for example, a vomica is near the surface of the lung, the pleura, already adherent to the parietes of the chest, thickens considerably; I have occasionally found it measure half an inch (Parisian). At the apex of the lung, where this process alone occurs, the walls of the thorax sink in, so as greatly to favour the shrivelling process. The corresponding bronchial tubes close up in like manner, and the healing of the cavity is accomplished. The remaining scar is either thick and roundish, or elongated and flat.

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[1] Judd (Lancet, 1838) has collected several of these cases, and shown their connexion with tubercular disease.

In the second instance the cavity is not obliterated, but remains open, simply losing the characters of the original disease. Its walls are moulded out of the aforesaid layer of shrivelled parenchyma, which now neither contains nor secretes tuberculous matter. These extinct vomicæ are frequently seen at the apex of the lungs, and might readily be taken for mere bronchial dilatations, were their real nature not disclosed by the relations of the surrounding pulmonary texture, by the coexistence of cretaceous and calcareous masses, and, above all, by the character of their membranaceous lining, which essentially differs from, and does not immediately unite with, the mucous membrane of the bronchial tubes. This internal coating for the most part adheres very firmly to the indurated walls of the cavity, and is either thick, reddened and velvety, or else pale, smooth and attenuate. It is often the seat of catarrhal or other inflammatory action, which communicates to it a roughness like that of a corroded surface, whilst the cavity fills with purulent matter. Here it is difficult to define the boundaries betwixt that and the bronchial mucous membrane, because the latter speedily assumes the same rugged aspect. If the cavity was originally extensive, several bronchial tubes enter it,—a circumstance that would prevent its being mistaken for bronchial dilatation. It very often happens that the bronchial tubes leading into such a cavity become closed—their orifices, contracted by the gradual shrivelling and obliteration of the adjunct texture, being eventually quite covered over with the membranous cicatrice. In exploring the air-passages after death, we often arrive at a dilated portion, beyond which the ulterior course of the bronchus is restricted to a whitish cord, and at length vanishes at the margin of a closed cavity. This last either contains gelatiniform mucus, or a thick pultaceous mass, of yellowish tinge, or it may be, cretaceous and calcareous concrements.[1]

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[1] See an interesting case in point, in Cerutti's work, pl. ii, fig. 6.

It is of importance to study in what manner not only the adjacent, but the more remote portions of the pulmonary texture are influenced by the curative processes above mentioned. The inflammatory exudation, upon which the aforesaid obliteration and shrivelling of the pulmonary cells depends, often involves the whole of the apex, if not the entire upper lobe of the lung, while at the same time the collective bronchial tubes, up to their very ends, degenerate into white, thread-like ramifications. The involved parenchyma of the lung is now converted into an almost cartilaginous mass, impervious to air, very scantily supplied with blood-vessels, and presenting, when cut, a smooth glistening surface. Obliterated pulmonary vessels, closed bronchi, cicatrices, and, finally, parenchyma drenched with plastic materials, are scarcely to be distinguished from each other, and the whole adheres very firmly, by means of the thickened, semi-cartilaginous pleura, to the sunken walls of the chest. A few cretaceous tubercles are still found scattered here and there throughout the hard, nearly homogeneous mass, which, below, merges sometimes gradually, but more often suddenly into the healthy texture.

A very remarkable fact is the extraordinary deposition of black pigment into the lungs, during the healing process. I have only found it wanting (or almost wanting) in the rare instances of the repair of tubercular mischief taking place through calcareous deposition, in youthful individuals. In older persons this melanotic accumulation is so constant and so considerable, that one might now and then entertain a doubt whether it be the cause of, or the sequel to the cure of phthisis. Not alone the parts immediately adjoining calcareous tubercles and cicatrized cavities, but, in like manner, the entire mass of those extensive indurations of the upper lobe before described, are found densely loaded with black pigment. Even the moist chalk-like residue of tubercular masses is often so imbued, as to exhibit a slate gray or a blueish-black tinge; nay, in the shrivelled cavities of nearly obliterated vomicæ it sometimes occurs as a smeary, blackened mass.

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[1] Some excellent illustrations of these points are figured by Reynaud (sur l'Oblitération des Bronches, Mém. de l'Acad. Roy. de Méd. vol. iv), and by Carswell (l. c. Fasc. Tubercle, pl. iv, fig. 5.)

In other instances,—chiefly, it would seem, where tubercle had not previously spread much, and in young individuals endued with greater vital energy, the pulmonary textures shrivel up, but without suffering induration or extensive obliteration. Here the scars appear rather streaky and elongated,—the cretaceous residue as isolated granules; the black pigment is uniformly, but less densely disseminated throughout the interstices of the air-cells. The cicatrized patches are surrounded with well-marked emphysema and with dilated bronchi, and the thorax is seldom found equably depressed. (See Emphysema and Bronchiectasis.)

In three cases I have observed that pre-existent *adhesions* at the apex of the lung begin to *loosen*, form lengthened threads or thin bands, and finally vanish altogether. The fact of their existence is afterwards attested by their semi-cartilaginous plates upon the surface of the top of the lung. These on different spots are, more or less, puckered in a radiate manner. Such are the appearances presented by the most perfect examples of recovery, which often allow not even a vestige of the residue of tubercles to be detected.

In children, the lungs are less prone to become the predominant seat of tubercles than other parts,—for example, the lymphatic glands and the bones. When deposited there in excess, however, they pass through the different stages of the destructive process rapidly,—although, in very young children, the disease is wont to follow a latent course, without revealing itself by any distinct signs during life. The lungs of scrofulous children, who have died of some other maladies, are often found to contain tubercles in the shape of grayish, very transparent, semi-fluid granules,—not, as in the adult, chiefly confined to the apex, but occurring equally in the inferior lobes. The bronchial glands are, at the same time, highly tuberculous. The relative frequency of tubercular hepatization in young children has been already discussed.

Billard met with 5 examples of tubercles in new-born infants, and Husson (Dict. de Méd., art. "Œuf") states, that he found softened tubercles in the lungs of a fœtus of 7 months; as did Kennedy in a still-born infant. (Dubl. Jour., May, 1839, p. 300.)

*Acute* tubercular affection occurs in childhood, and more particularly between the second and eighth years, as well as in adult age; but it is less apt to affect the lungs,—than the liver, the spleen, the kidneys, and, most of all, the brain and its membranes. It is altogether a very remarkable disease, puzzling in its symptoms, and liable to be mistaken for acute hydrocephalus.

The changes coexisting in other organs of individuals who die of phthisis of the lungs, are exceedingly numerous, and for the most part concur to show that the disease is dependent upon constitutional predisposition. *In the lungs,* besides the morbid conditions immediately connected with the tubercular deposit, we frequently observe various degrees of inflammation in the inferior lobes, usually more or less general œdema, both inflammatory, and passive; emphysema, especially of the upper lobes,—but, in lingering phthisis, generally diffused in front. The accession of the latter, seemingly, arrests the growth and progress of tubercular disease;—for, in the midst of thoroughly degenerate parts, we often meet with an emphysematous patch of lung exempt from tubercles. (See Emphysema.)

Bronchia, which terminate abruptly at the margin of a cavity, and present amid shrivelled texture, and in the neighbourhood of cicatrices, obliteration of their minuter twigs, but dilatation of their larger branches (see above), exhibit repletion of their caliber or infiltration of their tunics with tubercular matter, their mucous membrane being often reddened and puffy, appearing sometimes, especially where the tubes diverge, as if eroded, and at the same time coated with purulent mucus. As a general rule, this takes place only where they serve as outlets for the fluid effused into the morbid portions of lung. The mucous membrane of the trachea is, in like manner, more or less reddened, and that principally at the side of the lung most affected. It very often exhibits erosions, and even ulcers of greater depth. The larynx presents the same characters more strongly marked, (see Tubercular Disease of the Air-passages.) The bronchial glands are almost invariably implicated, and either pass through all the different phases of the tubercular disease, or else, in consequence of the inflammatory action pervading the pulmonary texture, appear swollen, softened, and of a dull gray, or black marbled hue. The glands of the neck and those situate along the trachea are apt to become affected in the same manner.

Towards the termination of phthisis there occurs, in the great majority of cases, pleural inflammation, productive of every variety of false membrane, of miliary tubercles upon the pleura itself and within its coagulable exudation; and, finally, of turbid, reddish, watery or purulent effusion. This inflammation differs, therefore, from that mere local process which gives rise to the adhesions, already described, at the apex of the lung.

The heart, externally, is quite devoid of fat, except in old persons, in whom the tubercular affection is of long standing, and complicated with certain morbid states of the liver. The reabsorption of the fat is sometimes incomplete, its former seat being still indicated by the presence of a reddish-yellow jelly. Both sides of the heart are filled with black coagulated blood, and where inflammatory action has prevailed within the respiratory organs towards the close of life, fibrinous clots, more or less extensive, are never wanting. G. Gluge (l. c. p. 81) constantly found the blood within the larger veins and the heart, in aggravated phthisis, contaminated with pus-globules, to which circumstance he is inclined to refer most of the peculiarities of hectic fever. A very remarkable fact was observed, first by Louis and afterwards by Bizot (Mém. de la Soc. d'Observ. vol. i); namely, that the aorta in pulmonary consumption, is found invariably—sometimes very considerably—coarctated, as ascertained by comparing the proportion of its caliber to that of the pulmonary artery in *this* disease with their mutual relations in a healthy subject. Atheromatous deposits within the arteries,—under other circumstances so frequent,—are not met with at all in young persons who die of tubercular phthisis, and in old ones more seldom than usual.

The liver almost always presents a variety of changes: its volume and absolute weight are augmented, and its blood-vessels are loaded; for the most part it is pallid,—of a rather yellowish hue, and very fatty; in many instances it assumes the nutmeg character. The proportion of bile is generally normal. The spleen is occasionally somewhat enlarged, and its texture more or less softened.

Shortly before decease, aphthæ appear upon the mucous membrane of the mouth, and may also extend to the œsophagus. The stomach is mostly enlarged, displaced in the direction of the mesogastric region, and acquires more of a vertical position, so that its great curvature, instead of forming its base, inclines rather to the left. The jejunum, but more particularly the latter portion of the ileum and the cæcum (more rarely the colon), present an extensive scene of disorganization and destruction,—the effects of tubercular action upon the intestinal glands and membranes. In older persons, more especially those who have been long consumptive, hæmorrhoidal tumours occur at the extremity of the rectum. To what extent these and fistula in ano are connected with tubercular phthisis, is not satisfactorily established. I have myself only in three instances seen phthisis associated with anal fistula. Wherever the alimentary canal becomes the seat of tubercular mischief, and sometimes even where it does not, the mesenteric glands are variously altered by the tubercular process.

The brain and spinal marrow, for the most part, remain natural,—but a considerable amount of serous effusion, resulting from the protracted death-struggle, and the impeded circulation, is almost uniformly accumulated beneath the arachnoid membrane, and within the cellular tissue of the pia-mater.

The external skin exhibits no general change of structure, although, towards the termination of the disease, it is wont to slough in the usual situations, from the patient being so long bedridden. The hairs on the scalp become dry and brittle, and stunted in growth; they fall out in considerable numbers, at first from the sides of the head. Where the disease is of long duration, the nails of the fingers and toes become incurvated and bulbous. Almost every particle of fat disappears from the subcutaneous cellular texture, and wherever else it is wont to lodge. In many instances, the cellular texture of the lower extremities becomes the seat of œdematous infiltration.

By a series of comparative experiments, carefully instituted, Clendinning (see Med. Chir. Rev. Jan. 1839) ascertained that, with the exception of the brain and heart, the absolute density of most organs, but more particularly of the lungs, liver, and spleen, is augmented in tubercular phthisis. It would, therefore, follow, that the enormous loss in weight generally sustained by the body, and amounting, on an average, to 48 lbs., is due solely to the disappearance of the adipose textures.

Louis (l. c.) has given the results of 120, Mohr (l. c. p. 90, &c.) of 25, and I have myself arranged, in tabular form, 100 cases,—all illustrative of the coexistence of tubercles in other organs, in phthisis. From these data it results, that all the other organs are not equally prone to deposit tubercles concurrently with the lungs. The bronchial glands are tuberculous in about one fourth of the cases; the mucous membrane of the larynx and trachea in about one twentieth only, and the cervical glands in like proportion. The intestinal canal, on the other hand, is affected in about one half the number of cases,—the mesenteric glands in more than one third. The mucous membrane of the stomach, which, according to Louis, never contains tubercles, does so, though very seldom. The serous membranes are often tuberculous, and in the following order of frequency: pleura—peritoneum—arachnoid—pericardium. The liver, spleen, urinary organs, brain, spinal marrow, and muscles, are only now and then engaged. Of the male organs of generation the testicles,—of the female, the mucous membrane of the Fallopian tubes, occasionally become the receptacle for considerable tubercular deposits.

Although the bones are very often the seat of tubercle, independently, they are very rarely affected in pulmonary phthisis. I have only witnessed this complication in nine instances. It would seem that tubercular caries acts as a sort of vent for carrying off the morbid matter,—whereby it is diverted from internal organs.[1]

The relation of tubercular phthisis to other diseases, as fully detailed by Rokitansky, is most interesting. (Oest. Jahrb. vol. xxvi, p. 23.) Louis (l. c.) had previously mentioned the rare occurrence of hypertrophied heart in tubercular phthisis; having, out of 112 cases, but thrice met with it, and that in a limited degree. Rokitansky, on the other hand, found that in 143 cases of hypertrophy of the heart, extinct tubercular disease of the lungs coexisted in 15 only. He correctly admits, however, that slight thickening of the walls of the *right* ventricle is by no means uncommon in phthisis. Of hypertrophy of the left ventricle, I have, myself, encountered but two examples out of 100 cases. Rokitansky observes, moreover, that in 108 instances of aneurism, tubercles were discoverable in but five. The association of tubercular phthisis with dysentery, with typhus, with cholera, with puerperal fever, with the various forms of cancer, and with hydatid development, is a rare exception. I have only twice seen hydatids conjointly with tubercles—once large hepatic cysts in a consumptive female. Further, Rokitansky found, in 50 cases of spinal incurvation, but three of pulmonary tubercles. In like manner, I have but thrice met with tubercle—extinct tubercle—amongst numerous instances of lateral curvature. To this may be added, that true apoplectic extravasations into different organs, even in young individuals, never occur during the progress of, and very seldom after recovery from tubercular phthisis; and, moreover, that variolous disease assails almost exclusively, non-tuberculous persons. The importance of these results, in a practical point of view, is self-evident, although it cannot be denied that some of them are directly opposed to preconceived opinions and old-established doctrines.

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[1] To children these relations, of course, do not apply.

## SECT. II.—TUBERCULOSIS OF THE BRONCHIAL GLANDS; BRONCHIAL GLANDULAR PHTHISIS.

The bronchial glands may, as stated in the foregoing chapter, become the seat of tubercle, either primarily, or in consequence of bygone tubercular disease of the lungs. The present remarks apply chiefly to the former, which, as an independent affection, has not heretofore obtained the consideration it deserves.[1]

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[1] Compare F. G. Becker (Diss. de Gland. thorac. lymphat. atque thymo); Andral

From the general character of tubercle, it is obvious that bronchial glandular phthisis may be conjoined with phthisis of the lungs and of other organs. The former, therefore, may, and often does, run its course without any tubercles becoming developed in the lungs,—or again, these organs may become implicated at any period of the disease, and be destroyed together with the glands,—or lastly, bronchio-glandular phthisis may subside: nevertheless the tubercular tendency is not extinguished, and leads in the sequel to pulmonary consumption, whereby the disease of the glands is again called into activity. This is essentially, and almost exclusively, a disease of childhood, as is, in a great measure, the case with scrofulo- tubercular disease of the lymphatic glands generally. Although, therefore, in advanced, or even in old age, the bronchial glands are found to have undergone the changes peculiar to tuberculous deposit, these are generally of long anterior date,—virtually, the remains of cured bronchial phthisis, which circumstances had once more developed. Bronchial glandular phthisis, first originating late in life, is, on the contrary, rare.[1] and almost always the mere accompaniment of lingering and protracted pulmonary phthisis. It most commonly sets in between the first and second dentition, and finishes its course with the advent of puberty, although its consequences are sometimes manifest beyond this period.

The course of bronchial glandular phthisis is almost always chronic, and the destruction and shrivelling of the glands, principally involved, proceeds so gradually as to allow the organism time to compensate for the damage. Hence it readily admits of reparation; nor is it in itself perilous, although accidental circumstances may render it fatal. In the dead body, therefore, the morbid changes peculiar to this form of phthisis are only to be studied in the final stage, unless where death has resulted either from intercurrent disease, or from certain con-

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(Clin. Méd. vol. iv, p. 248.) The disease in children has been described by Leblond (Diss. sur une Espèce de Phthisie particulière aux Enfans, Paris, 1824); Berton (Traité des Maladies des Enfans, 1837); Rilliet and Barthez (de la Tuberculization des Ganglions bronchiques, Arch. Gén. Janv. 1840.) A portion of my observations, relative to children and adults, has been published by Kerstein (Observat. quædam de Phthisi bronchiali. De Lips. 1842.)

[1] By this is understood *true* bronchial glandular phthisis,—not the symptomatic implication of the bronchial glands in pulmonary phthisis.

tingencies about to be specified. Nevertheless, it is expedient to take every occasion of making ourselves familiar with those earlier changes, as offering the only satisfactory means of accounting for a variety of symptoms witnessed in young persons up to and beyond the age of puberty.

As in the lungs so in the bronchial glands, tubercular development follows a particular course. The glands at the bifurcation of the bronchi are earliest attacked, and also first pass through the several morbid stages. From thence the morbid process generally diverges in three distinct directions, extending first to the lymphatic glands which follow the ramifications of the bronchial tubes deep into the pulmonary texture; secondly, to those situate between the pericardium and the lungs, and along the œsophagus in the posterior mediastinum; and lastly, to those which accompany the large vessels in the anterior mediastinum, and pass from thence to the trachea and to the cervical plexus. Only where the tubercle is primarily seated in the mesenteric glands, does it appear to advance along those glands which follow the course of the œsophagus. passing from thence to the cervical plexus. In some instances, the tubercle probably originates in the glands of the neck. An opportunity seldom offers for examining tubercles in the nascent state. They then present the appearance of gray or yellow granules, up to the size of millet seeds, and occupy partly the centre and partly the circumference of the glands. The latter are always somewhat enlarged, either softened or hardened, and of a colour inclining to gray. In a short time they become so infiltrated with tubercle as to constitute a yellowish white friable substance, in which no vestige of their pristine texture is discernible. Under these circumstances they enlarge to an extraordinary degree,—those about the bifurcation of the trachea attaining the size of a pigeon's egg or more,—the remainder, especially within the lung itself, that of a hazel-nut,—in decreasing progression, according to their remoteness from the part originally affected. If before but loosely attached in the midst of cellular tissue, they now, through its medium, coalesce very firmly with neighbouring parts, more especially with the bronchi, acquiring, from the lardaceously hardened cellular texture, a solid isolating envelop.

It is very remarkable, that glandular enlargements, however extensive and numerous, comparatively seldom produce very marked symptoms of the compression of nerves and blood-vessels,—although great and important trunks run in the vicinity. Tubercular disease of the bronchial glands having, however, been hitherto little noticed, such symptoms may probably have been interpreted otherwise. Hugh Ley (on Laryngismus Stridulus, &c., London, 1836), after repeated observations, was induced to refer the spasmodic asthma of children to the pressure of swollen glands upon the eighth pair, and especially its reflected twigs. But, although the neurilemma is found in one case thickened,—in another flattened by compression,—and uniformly adherent to the tumours,—still nervous filaments can always be detected, by careful dissection, traversing such localities unchanged. The arteries appear to experience no great pressure, even where the lymphatic glands are most firmly attached to their external cellular coat. Not so the veins, because their yielding tunics, and the inferior pressure of their contained column of blood, offer less resistance. The bronchial tubes, more especially within the lungs, are seldom compressed, owing, on the one hand, to their being kept on the stretch by their cartilaginous framework,—on the other, to the more yielding substance of the lung allowing ample space for the tumour to expand. Sometimes, however, the pressure overcomes all resistance, and intercepts more or less the access of air to the lungs, as shown by Reynaud in the instance of a monkey (Mém. de l'Acad. vol. iv, p. 164), and by Andral in that of several human beings.

The tuberculous mass accumulated within the bronchial glands may long continue in the condition above described; for the most part, however, it sooner or later undergoes softening. Where this proceeds from the centre, one or more little excavations are found, containing a gritty, purulent fluid. In this case the whole process is very tedious. Where, on the other hand, the softening commences at the circumference, the filamentous sheaths of the glands are found highly vascular and puffy,—ultimately constituting a mere cyst around the thick, yellowish, tuberculo-purulent fluid. The tumour has now attained its utmost magnitude, and from this moment collapses, its contents either becoming gradually absorbed, or else escaping through a passage of their own making.

Absorption here proceeds very slowly, being in a great measure carried on by the external vascular coat of the tumour, the inside of which is of a lively red, and of a velvety aspect. Like a vomica, for the most part it possesses an unorganized membranaceous lining, fabricated out of thickened pus and tubercle. The cavity thus formed does not, however, like the pulmonary excavation, continue to enlarge; its walls receive no additional deposition of tubercle, and rather seem to act as a shield; indeed, no instance of the reduction of two adjacent glands into one common excavation, has ever occurred within my experience. On the contrary, in proportion as its fluid contents are absorbed, the cyst closes in upon the more consistent, pap-like residue, which, lessening by degrees, acquires a mealy and cretaceous character, and eventually becomes converted into a calcareous concrement of stony hardness. Such products are frequently met with, even in youthful individuals, in the place of one or other of the bronchial glands, more especially at the bifurcation of the trachea. (Becker, l. c. p. 21.) This morbid process does not, however, extend equably to all glands, but rather observes the order of sequence which the original tubercle-formation had taken.

Wherever the tuberculous mass has remained fluid till long after puberty, or the disease has arisen at a later period, black pigment becomes deposited, both in the unsoftened portion of the tumour, and in the pap-like mass. With the latter it becomes so intimately combined, as ultimately to constitute an uniform, black, smeary pulp. In such instances, the tumour, whilst gradually contracting in dimensions, retains the same soft character for years. Sometimes separate calcareous nuclei are found in the midst of the blackened mass. This deposition of pigment within glands, totally degenerate, and partially destroyed, is the more remarkable, as furnishing a proof of connexion with the lymphatic vessels. It can scarcely be reckoned an immediate secretion from the glandular cyst, and *necessarily* concerned with tubercular cicatrization,—because this process in other organs, (the lungs excepted,) and especially in lymphatic glands, is scarcely attended with any deposition of black pigment.

Softened tubercle very frequently escapes by perforating the bronchial tubes. These perforations take place most commonly from without inwards, (rarely in the inverse direction) commencing in ulceration of the bronchial mucous membrane, which eventually burrows into the tuberculous gland. The tuberculo-purulent fluid now commonly bursts through the bronchus, determining an irregular, and more or less extensive opening, through which it gradually escapes. Years are then required for perfect recovery,—which is brought about, either by the aperture gradually healing to a point, whilst the subjacent gland shrivels away, or else by its closing ere the contents of the gland are entirely voided,—the remainder subsequently passing through the several phases described, until it undergoes calcareous metamorphosis. Occasionally a renewal of irritation restores the communication at a later period, and little calcareous concrements come away one by one. The blackish and deep scars, thus formed, are very frequently encountered upon the mucous membrane of the bronchia, and their real origin is easily revealed by making a vertical section, whereby the subjacent shrivelled gland is simultaneously divided When the above process affects glands deeply situate within the lungs, where the bronchi are no longer upheld by a perfect cartilaginous framework, the perforated canal contracts considerably after healing, and this narrowing is the more conspicuous, inasmuch as within the lungs the glands are always seated at the acute angle of the bronchial divisions. For the same reason it is usual for two canals to be perforated at once. Sometimes these perforations of the bronchial tubes are repaired in a different manner; the opening progressively enlarges, until at length an extensive excavation occupies one side of the tube. The continuity is ultimately restored through cohesion of the glandular walls, the loss of substance being compensated for by the cavity filling with a fibro-cartilaginous mass. This would, however, appear to be an extremely slow process, for I have frequently met with capacious openings of the kind in the larger bronchial tubes of very old persons. The sustained irritation about these exulcerated patches, causes the bronchial cartilages to ossify, and afterwards to project, as naked splinters, beyond the brink of the cavity.

In some few instances softened tubercle escapes into the parenchyma of the lung, giving rise to cavities which closely resemble those resulting from pulmonary tubercle. They are indeed to be distinguished from them only by their invariably occurring near the spot where the great vessels and bronchi enter the lungs, by their direct communication with tuberculous glands, by their being, for the most part, limited in size, and smooth on the inside, and by the circumstance that the bronchial tubes do not enter them straight, that is, with the whole ring of their corroded extremity, but open into them, only through a lateral and often very narrow orifice. Where the parenchyma of the lung is, at the same time, the seat of tubercular degeneration, discrimination is very difficult and of no practical importance.

*Perforation into the pleural cavity* happens still more rarely, and is, indeed, only possible where the softened glandular masses are situate immediately under the pleura, at the interlobular divisions. Their escape into the above-named cavity generally determines a more or less violent pleurisy, not necessarily fatal, except in the extraordinary coincidence of the same gland having perforated a bronchial tube, so as to induce pneumothorax;—a catastrophe witnessed both by Berton and by Rilliet and Barthez.

*Perforation of the œsophagus* is less rare. Berton and Leblond have each seen an example in children. Here the pus accumulated within the glandular tumour is evacuated by degrees, and, it would appear, in occasional gushes, when retching and coughing favour its expulsion. In this manner a very large quantity of tuberculo-purulent fluid is sometimes discharged at one time. When the lymphatic gland communicates with the œsophagus alone, perfect reparation ensues, the emptied tumour collapsing, firmly coalescing with the œsophagus, and thus contributing to establish a deep cicatrix. I have met with five examples of scars of this nature in aged persons, the base having, in every instance, been formed out of the hard, blackish remains of a gland. Simultaneous perforation both of the œsophagus and of the trachea, probably always proves fatal, through food entering the air-passages. Such an accident may befal adults. Syme (Edinb. Journal, vol. xliv, p. 113), records the case of a man, aged forty-five, in whom a tuberculous bronchial gland was found to communicate at once with the trachea, the œsophagus, and the pericardium.

It is possible that tuberculous glandular tumours often bring life into jeopardy by becoming intimately attached to the parietes of the great vessels. Shrivelled glands are at least often found so thoroughly incorporated with the thin membranes of the pulmonary artery, that its internal coat alone admits of separation in an uninjured state. Of actual perforation, however, but two examples are known, both in children. A main bronchial trunk and a large branch of the pulmonary artery were simultaneously penetrated by a tuberculous gland; death took place instantaneously, under profuse hemorrhage.

In children, in whom alone the disease occurs independently, the lymphatic glands of other parts, especially of the mesentery, often afford the sole evidence of concurrent disease. The lungs are, in particular, often quite free from tubercle. Papavoine remarks, that of forty-nine children affected with tubercle of the bronchial glands, but thirty-eight had tubercles in the lungs. Rilliet and Barthez never found other organs in children tuberculous, without the bronchial glands being preeminently so. Still bronchial glandular phthisis in children very often leads to acute general tubercular disease, and then the lungs, the liver, the spleen, the kidneys, and most of the serous membranes, are all found to contain recent tubercles. In adults, on the contrary, in whom either recent, or the remains of by-gone, bronchial glandular phthisis may be discovered, tubercular disease of the lungs always predominates, and is in itself mortal.

## SECT. III.—TUBERCULOSIS OF THE AIR-PASSAGES. LARYNGEAL AND TRACHEAL PHTHISIS.

*Ulceration of the larynx and trachea* is, in the majority of cases, of a decidedly tubercular character. Upon this point Albers, Trousseau and Belloc, and Barth,[1] who have specially studied the subject, are fully agreed. Nor am I aware of any instance of those organs running through a course of tubercular disease independently of pulmonary phthisis. The cases recorded as such by Trousseau and Belloc, were in reality either of a syphilitic or of a carcinomatous character, or else the consequence of perichondritis, of polypous growths, or of simple catarrhal irritation, and had no connexion with genuine tubercle.

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[1] F. J. H. Albers (Die Pathologie und Therapie der Kehlkopfskrankheiten, Leipzig 1829; and his Beyträge zur Pathol. Anat.); Trousseau and Belloc (Phthisie laryngée, Mém. de l'Acad. Roy. de Méd. vol. vi, 1837); Barth (Arch. Gén. de Méd. Juin 1839.)

The relation of laryngeal and tracheal to pulmonary phthisis varies greatly. In most cases pulmonary affection pre-exists, the larynx and trachea becoming merely implicated during its progress. Sometimes the disease begins in the three organs all at once, and predominates in one or other, in the sequel. The lung is generally the part that principally suffers, and the instances are rare in which pulmonary consumption lingers, remains stationary, or retrogrades, while the laryngeal phthisis is pre-eminently developed, and of itself the cause of death. It is believed that the affection sometimes assails the larynx (never the trachea) in the first instance, and afterwards extends to the lungs. This fact is, however, not thoroughly made out; perhaps it may happen where the tubercular diathesis quickens, after a long period of abeyance. Where laryngeal disease coexists with the pulmonary affection, the latter is apt to be overlooked, unless a very scrutinous investigation be made. This is obvious from the anatomical relations of the two,—laryngeal disease being far more readily, and in a far greater degree, productive of disturbance to the respiratory function, than incipient pulmonary phthisis, and at the same time more striking, from its influence upon the voice, and from its more painful character. Stokes, therefore, judiciously recommends the closest attention to be paid to the condition of the lungs, during the progress of the other affection.

It is a very remarkable fact, that tubercular ulceration of the larynx and of the trachea occurs principally between the twentieth and thirty-fifth years; in advanced age it is far less frequent, although even then it may cause serious damage. It is rarely observed before puberty. Portal alone records one example in a girl of eight years. A marked difference presents itself relatively to sex, males being more subject to this kind of ulceration in the proportion of two to one. This is all confirmatory of the law before laid down, namely, that organs are most prone to the development of tubercle, while at the height of functional vigour.

Tuberculous ulcers are not equally frequent in all parts of the air-passages. According to Bayle, Andral, and Barth, their frequency progressively diminishes from above downwards; and with this accords my own experience. Louis, indeed, mentions that out of 102 phthisical patients, he found the epiglottis 18 times, the larynx 22 times, the trachea 31 times, and the bronchi 7 times affected. This, however, is only a seeming discrepancy, since he does not recognize any substantial difference between the manifold forms of ulcer and erosion. Upon the whole, it may be asserted that true tubercular lesions have their seat mainly in the larynx, and are seldom met with in the epiglottis and trachea, exclusively of the larynx. Bronchial ulceration is, for the most part, immediately connected with phthisis of the bronchial glands, or of the pulmonary texture.

With a view to obtain a more perfect acquaintance with the affections of the air-passages in phthisis, it is important to distinguish several essentially different forms. Some are not of tubercular origin, but mere simple, superficial erosions of the mucous surface,—or softenings and disorganizations of the muciparous glands,—the result of irritation.

*Simple erosions of the mucous membrane* are associated either with a slight inflammatory condition of the mucous membrane,—or else with ulcers. They are sometimes seen only in certain localities, as the inferior surface of the epiglottis, or the posterior surface of the trachea; frequently, however, the entire surface of the mucous membrane is beset with them. They are almost always of one uniform size, about the circumference of a lentil, though rather oval-shaped,—except where several have coalesced, which frequently happens in the bronchi, and imparts to the whole inner surface an eroded aspect. The erosions always remain superficial, the upper layer of the mucous membrane—probably the epithelium—being alone engaged. Hence they are not always detected at first sight. The base is for the most part smooth and pallid,—seldom faintly reddened,—frequently covered with a thin, whitish, and very soft pellicle, much resembling cast-off epithelium. Their edges are sharp and linear, whitish or reddened, or scarcely discernible. The surrounding mucous membrane is very often quite pale, and only now and then exhibits a red areola. These erosions are obviously the sequence of superficial irritation of the mucous membrane; and, as they are principally met with in parts which come in contact with tuberculous matter expectorated from the lung, they not improbably owe their existence to this source.

Together with these erosions, which appear to constitute the most frequent lesion of the mucous membrane in phthisis, the muciparous glands are often found affected, quite independently of tubercle. They appear, in the trachea, more particularly between its rings, in the form of white granules, about as big as mustard seeds, in the midst of a tumefied and strongly-injected patch of mucous membrane. They are, for the most part, highly softened, so that their covering ultimately disappears,—leaving smooth, excavated pits, no bigger than pins' heads, in the place of the granules. These changes are probably the consequence of catarrh grafted on tubercle.

*Real tuberculous ulcers* present a notable difference from the above. Louis, indeed, disputes their tubercular nature, and reckons them mere products of chronic inflammation, kept up by the irritant quality of the sputa in phthisis. Andral does not express himself very distinctly on the subject. Nevertheless, the generally well-marked tubercular granulations within these ulcers, and the determinate character of their microscopic elements, remove all doubt as to their true origin. They are most frequent in the larynx,—in its upper half, and at the posterior or anterior angle of the vocal chords,—but often occupy the whole inner surface of the organ. In many instances they extend to the lower side of the epiglottis,—sometimes beyond it, to the root of the tongue, even to the pharynx, and now and then to the entrance of the œsophagus. In the trachea they are less frequent than in the larynx, and chiefly confined to its posterior membranous walls. They are most rare of all in the bronchi.

Tuberculous ulcers are always much larger than those before mentioned; they are irregular in shape, and prone to deepen soon. Their base is rough, as if gnawed, and is generally made up of lardaceous thickening and hardening of the submucous texture, wherein are frequently discernible the little grayish, or yellowish-white granules, generally characteristic of tubercular formation. Their edges are prominent and abrupt, yet in the highest degree irregular and angular. The sores, when recent, are generally more uniformly circular, having a smooth, pallid base, neither indurated nor granulated, but with regular, flat, and soft edges, derived from mucous membrane, which seems but little changed. In the latter case their circumference is not reddened,—rather, indeed, more pale than natural; when, however, they are of chronic origin, it is mostly grayish, or of a deep red. Tuberculous ulcers invariably tend to spread both superficially and in depth, through the injurious influence exercised upon the implicated textures by the continual fresh deposition and softening of tubercle at their margins and base.

The opportunity of observing these ulcers at the outset is rare; they would, however, appear to originate in various ways. Tubercle commonly accumulates within the capsules of the muciparous glands, elevating the latter into little eminences, and ultimately, when the softening process is completed, leaving corresponding ulcers in their stead. In many cases, again, tubercles form, in the first instance, within the mucous membrane, the process being probably as follows: instead of normal cells, tubercle-cells form beneath the epithelium-cells, crowd together, in part reach the surface and are shed, in part irritate the contiguous textures, producing, first, loss of substance, and afterwards ulcers. In other cases, again, tubercle is from the outset deposited within textures still more deeply seated.

When tubercular destruction does not originate at a depth from the surface, but only gradually descends, the subjacent textures undergo manifold changes. The most remarkable of these affect the cartilages and their perichondrium, and are much more frequent and conspicuous in the larynx than in the trachea. The irritative inflammation constantly kept up in the vicinity of ulceration, determines, sooner or later, a deposition of phosphate and carbonate of lime betwixt the perichondrium and the cartilages, at first upon their inner, afterwards upon their outer surface. This calcareous deposit primarily appears in the shape of irregular, rough little scales, which progressively enlarge, and keep encroaching upon the cartilage, until it is altogether destroyed,—one compact bony plate having assumed its place. This variety of ossification, which essentially differs from that incident to old age, was first accurately pointed out and considered in its intimate relation to laryngeal phthisis by Trousseau and Belloc. It is hardly ever quite complete, but here and there is reduced to a thin incrustation; sometimes the perichondrium is converted into a calcareous crust. The change in question most frequently affects the posterior portion of the cricoid cartilage,—next in frequency the thyroid,—far more rarely the arytenoid cartilages, whilst of ossification of the epiglottis but four instances are known; they are recorded by Albers (Gräfe and Walther's Journal, vol. xxix, p. 20). I have frequently found the ring of the trachea and bronchi thus transformed. (See above.)

In the progress of tubercular destruction, the ossified cartilages are themselves liable to exulceration, both from without and (as Albers accurately states) from within. In the former case, minute ulcers very speedily penetrate deeply, so that at length their base is constituted by the denuded perichondrium; in these, tubercle becomes deposited, softens, and ultimately exposes the subjacent ossification, which then gradually crumbles away. In the second, and by far rarer case, tubercle is generated immediately within the cellular spaces of the ossified cartilage, and in softening determines internal caries. In this way a little abscess is formed, which elevates the superincumbent soft parts, and eventually opens through them into the larynx, to permit the escape of pus and carious fragments. This latter process occurs only in elderly persons, in whom the cartilages had become ossified in the ordinary manner, prior to the date of the tubercular affection. It is not uncommon for a large-sized and profound ulcer to expose and destroy a considerable extent of perichondrium, whereby a largish fragment of ossified cartilage dies, and becomes exfoliated. Such fragments are often found, more or less loosened, within the ulcers; the greater portion of the thyroid or cricoid cartilage is said to have sometimes come away in this manner. The process is more commonly limited to smaller fragments, and it may be doubted whether, in these cases, very extensive necrosis be not rather the sequel of perichondritis. During great debility, it may happen that the detached portions are not ejected but become impacted in the glottis, causing instant death by suffocation.

The disease very often runs a rapid course, the ulcers reaching the cartilages ere the latter have become ossified. Caries, or necrosis, then follows, under the conditions above specified.

The first is most frequently perceived in the arytenoid cartilages, which never ossify. These cartilages are sometimes destroyed, from the apex to the base, by progressive ulceration, and whatever portion of them remains, is firmly cased within the lardaceously thickened perichondrium. In like manner cartilages often become denuded, before they have had time either to ossify or be removed by ulceration. Exposed cartilages projecting into a tuberculous ulcer presently soften down into a caseous mass. They are not detached in large lamellæ, but liquefy gradually in the surrounding purulent fluid.

In phthisical subjects I have more than once found tubercle deposited in the space intervening between the cricoid and one or both arytenoid cartilages (compare Andral, l. c. vol. iv, p. 191), and this, moreover, in cases where no true ulcers were observed in the larynx. The result is an altered position of the arytenoid cartilages, with immobility of the vocal chords. The voice is either deprived of tone, or its production is difficult and painful. Up to this point cadaveric inspection affords no adequate explanation of the above symptoms. By and by, the tubercle softens at the deepest part, and breaks through the mucous membrane, engendering ulcers which first involve the posterior point of insertion of the vocal chords, subsequently overspread them more widely, and ultimately reach Morgagni's ventricles.

The epiglottis, whose structure is rather *fibro*-cartilaginous, akin to the elastic tissues, suffers no change of a carious or necrotic character. It is, however, not unfrequently destroyed by ulceration. The ulcers always commence upon its inferior surface, from whence, however, they may pass round its margins to the front, imparting to these an angular and notched appearance. The whole epiglottis thus becomes thickened and unyielding; it is firmly bound by means of lardaceous, degenerate cellular texture, to the margin of the thyroid cartilage, is rendered less easily separable from the hyoid bone, and is closely attached to the root of the tongue. Sometimes it is found half, or even wholly destroyed, when filamentous, softened, and discoloured shreds alone overhang the glottis, or the entire organ appears, as it were, smoothly cut away. Whenever the upper surface is involved, ulcers, more or less extensive, are found upon the root of the tongue and in the pharynx.

The varied and extensive destruction affecting every part and texture that compose the larynx, produces in it notable tumefaction, and an alteration of shape discernible even from without,—its sharply-circumscribed outline being transformed into a soft, roundish, painful prominence. It is, for the most part, almost motionless, the ascent and descent, perceptible during speaking and respiration in other diseases attended with dyspnœa, being here no longer observable.

Sometimes tubercular devastation oversteps the boundaries of the larynx, chiefly, however, when the ulcers assail the upper portion of the angle of the thyroid cartilage. This soon yields, whereupon tubercular development and softening make further progress upon the outer surface of the larynx, engendering fistulous canals between the muscles, and purulent infiltration of the cellular texture. In these rare instances the destructive process, ere long, reaches the external skin, loosening it to a considerable extent, and causing it to slough and give way at various points. Andral relates an instance of a fistula of this kind lasting for nearly a twelvemonth. (Vol. iv, p. 189.) Meanwhile the cellular texture either perishes through suppuration, or occurs in mortified shreds in the midst of fistulous passages. The muscles and ligaments appear as if dissected; they are invested with a thin tuberculo-purulent membrane—are in a high degree softened—either pale as though macerated, or slate-gray—almost blackish; sometimes numerous miliary tubercles are discernible among their fibres. The cartilages project into the slough, denuded, softened, discoloured, and in a state of leathery shrivelling. If they have been previously ossified, single carious fragments may be found within the fistulous passages; small and even largish necrosed fragments have been known to come away through the cutaneous openings.

Examples occur, though more rare, of ulcers breaking through the posterior wall of the larynx, causing suppuration in the cellular texture interposed between the air-passages and the œsophagus, and eventually perforating the latter.[1]

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[1] Sachse (l. c. p. 193, et seq.) This casualty appears more common in perichondritis and syphilitic ulcers. (See Barth. Arch. Gén. 3$^{me.}$ série, vol. ii, p. 282.)

In the trachea, tuberculous ulcers are incomparably less frequent, but behave, in other respects, precisely like those of the larynx. They are most frequent upon its posterior wall, where they may, in like manner, burrow, and eventually perforate the œsophagus. (Andral, vol. iii, p. 169.) The anterior wall may become similarly ulcerated, giving rise to ossification and partial destruction of the cartilaginous rings, which then project in a broken state into the ulcers. I have observed this kind of ulcer at the upper extremity of the trachea, just below the cricoid cartilage. It advanced to beneath the external skin.

In the bronchial tubes these ulcers are still more rare. They are, for the most part, inconsiderable; Carswell has, however, given a figured representation of a bronchus firmly attached to a main branch of the pulmonary artery, with the walls of both perforated by a tuberculous ulcer. (Fasc. Hemorrhage, vol. iii, fig. 5.) Sudden death ensued, under violent hemorrhage. In general, the bronchial mucous membrane is but superficially eroded, and allows the little muciparous glands to be seen as white points, upon a deep red ground. This appearance is most palpable in the bronchial tubes of the more diseased lung. Although cicatrices of various kinds are frequently met with, we possess no positive information concerning the reparation of tuberculous ulcers within the air-passages.

The changes peculiar to laryngeal and tracheal phthisis, are often conjoined with the inflammatory diseases of the air-passages described in the preceding chapters. The most common of these complications is œdema of the glottis, and of the upper margins of the larynx, leading to the epiglottis. Inflammation of the mucous membrane, with thickening both of that and of the submucous tissue, is by no means rare. Andral has seen membranaceous effusion upon the inner surface of the larynx and trachea, though not of any great extent. It occasionally happens, that tuberculous ulcers which reach the cartilages, produce rapidly fatal inflammation of the perichondrium. Aphthous shedding of the epithelium in the air-passages, mouth, and fauces, here again sometimes directly precedes death.

After what has been said respecting the relation of pulmonary to laryngeal and tracheal phthisis, it may be inferred that the changes accompanying the one, will be observed also in the others. Ulceration of parts adjacent to the larynx,—as the pharynx, the œsophagus, the root of the tongue, has been already discussed. I once found the tongue studded from the root to the point with little ulcers, evidently tuberculous. The lymphatic glands, situate along the trachea and near the larynx, are very frequently, and, where the destruction is at all considerable, invariably swollen, reddened, and hard,—or else grayish and softened,—being, for the most part, secondarily tuberculous.

*Appendix on congenital fistula of the throat or neck.* Fistulæ resulting from tubercular destruction are not to be confounded with a congenital affection, first described by Dzondi,[1] and subsequently by Ascherson,[2] the real pathological import of which has, however, owing to the few cases examined after death, not as yet been thoroughly made out. Two varieties may be distinguished: one where the fistulous passage seems to communicate with the trachea; the other, where it opens into the pharynx or œsophagus.[3] The external orifice of the fistula was in every instance very narrow, surrounded with an elevated red areola, and not unlike an acne varus. It was sometimes on the right, sometimes on the left side, and twice at the centre of the throat or neck; in the first variety, in the proximity of the larynx, in the second, somewhat above the sternal extremities of the clavicle. The fistulous channel appeared to widen at a certain depth, and did not pursue a straight course.

Ascherson refers this condition to imperfect closure of the branchial fissures, described by Rathke, as existing in the embryo. In every instance the defect was congenital; it often recurred in the same family, seemed hereditary, and not unfrequently persisted during life. In thirteen cases the subjects were females; in three, males.

## GLANDERS IN THE HUMAN SUBJECT.

Of late years various pathologists have recorded instances where persons tending horses labouring under glanders, have been attacked with a very similar disease. Although many medical men of eminence, more especially members of the Parisian Academy, have questioned the fact, the infection has been proved, almost to demonstration. First, the disease has been only known to assail individuals previously in contact with glandered horses; secondly, the organic changes about to be described resemble those of genuine glanders in every respect, making allowance for the different organization of the horse and of man; and, thirdly, direct experiments have shown, that solidungulous animals may be inoculated with glanders, by means of fluid taken from the parts so diseased in the human subject. (Travers, Coleman, Rayer, Nonat.) The precise conditions under which the transmission takes place, belong more properly to the general doctrines of contagion, the obscurity of which, in reference to certain diseases, is well exemplified by the history of the one before us.

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[1] De fistulis tracheæ congenitis. 1829.

[2] Diss. de Fist. colli congenit. Berol. 1832.

[3] Baereus, Mittheil. Rigaischer Aerzte, vol. i, p. 5.

The first authentic case of glanders in man was that recorded by Schilling, in 'Rust's Magazine,' in the year 1821 (vol. xi, Fasc. 3, p. 480). Similar observations were communicated, almost as early, by Muscroft, in the 'Edinb. Med. and Surg. Journal' (vol. xviii, p. 321); and soon afterwards by others, both in Germany and England. The attention of the medical profession was, however, more generally awakened to the subject by an essay "On glanders in the human subject," by Elliotson, published in the 'Medico-chirurgical Transactions' (vol. xvi); and especially by Rayer's full and elaborate researches (Mém. de l'Acad. de Méd., vol. vi, 1837, p. 625), wherein all the cases of acute and of chronic glanders and farcy, then known, are skilfully analysed.

Glanders has its principal seat, both in man and horse, on the one hand, in the nostrils, and adjacent cavities, from whence it descends to the larynx, and even to the more distant respiratory organs,—on the other hand, on various parts of the cutis.[1] The affection of the Schneiderian membrane appears to set in rather early, between the fourth and the eleventh day, judging from the dribbling of the nostrils,—a symptom probably often overlooked at first. The eruption on the skin does not break out before the twelfth day of the disease.

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[1] It may, perhaps, be blamed, that glanders should here be classed with diseases of the respiratory organs, rather than with those of the skin. But the eruptions, deposits, and abscesses, described by authors, do not belong exclusively to the skin, whilst they sometimes depend, as we shall afterwards see, upon the manner in which the poison has been introduced. Moreover, the skin-affection is, in many cases, decidedly due to the simultaneous transmission of farcy, and it generally makes its appearance comparatively late. In the horse, glanders originally and chiefly affects the nostrils and air-passages.

In every case in which the Schneiderian membrane *was* examined,—a point too often neglected, unfortunately,—it was found more or less extensively swollen, inflamed, and destroyed by sloughing or ulceration, so that the cartilages and bones sometimes lay exposed. Adjacent to the mucous membrane, there was serous or purulent infiltration, and occasionally bloody ecchymosis. Now and then it was the seat of various kinds of eruptions, assuming the form either of lentil-sized pustules, grouped together upon a swollen and red, inflamed ground, and containing a yellowish mass, resembling boiled white of egg, intermingled with pus-globules, or of numerous scattered vesicles filled with pus; or again, of a congeries of little tuberculate bodies, varying in size from that of a pin's head to that of a lentil. The same appearances were traced into the frontal sinuses, into the cells of the ethmoid bone, and into the maxillary and sphenoid sinuses. The entire surface of the mucous membrane secreted a thin ichorous fluid, or was covered with a tenacious discoloured mucus, beneath which it was rough, as if both eroded and swollen. Sometimes the mischief stopped short at the posterior opening of the nostrils. For the most part, however, it advanced beyond, producing analogous phenomena on the arch of the palate, upon the tonsils, less frequently upon the tongue, and at other parts of the mouth, at the entrance to the Eustachian tubes, and in the pharynx. In almost all the cases minutely examined, the epiglottis and its vicinity, as well as the upper portion of the larynx, and once or twice the trachea, were found affected. The larynx exhibited precisely the same alterations as the nostrils, to wit, inflammatory redness, ulceration, and pustular development. In the trachea these were restricted to separate patches of inflammation, to formation of tiny vesicles, and to a layer of tenacious puriform mucus.

In several instances the lungs were sound, whilst in others they were unusually loaded with blood, or affected with diffuse or lobular inflammation; or, again, they presented, subjacent to the pleura, superficial abscesses or ecchymoses. The mucous membrane of the bronchial tubes had, generally, a florid appearance.

The mucous membrane is prone in this disease to various eruptions, resembling varicella, ecthyma, or small furuncles. Two distinct forms are cognizable: the one pustular, being the more constant, the other vesicular, present, if not in all, at least in the majority of cases. The pustules come out upon the anterior surface of the body, especially on the face,—next in frequency upon the chest and the extremities. They attain the size of a split-pea and upwards, and occur, either singly or in groups, upon an indurated base, surrounded by a dirty-yellow, livid, or dark-red areola. At first, they contain merely a plastic, yellowish-white substance, not only deposited beneath the epidermis, but also imbedded, as it were, in an opening in the corium. As the disease advances, the contents of these pustules soften and become transformed into pus, which perforates the summit of the pustules, and dries to a scab on exposure to the air. Underneath this scab, suppuration for the most part goes on, until an ulcer is produced. The pustules by no means break out simultaneously or equably, but are found scattered in every different stage of development. The vesicular eruption predominates in situations where the external skin is disposed in folds or wrinkles, as upon the ears, between the fingers, upon the genital organs, and the like; but, at a later period, also, in other localities. The vesicles are of various sizes: some attain fully the diameter of a half-crown piece; their circumference is livid, almost black; they contain a serous, dingy red, or a thin puriform fluid. They soon burst, or are rubbed off, and leave a gangrenous, copiously secreting spot, at whose borders smaller vesicles of the same type continue to form.

Suppuration sometimes occurs beneath the skin, betwixt the muscles, and both beneath and within the periosteum. Here, likewise, different grades of morbid deposition are perceptible. In a few instances, more or less extensive ecchymoses are present; in others, effusion of limpid or turbid serosity; in others, again, a jelly-like mass. For the most part, however, there are different sized abscesses, either terminating abruptly in healthy structure, or else surrounded by sero-purulent infiltration within softened texture. The pus itself often appears laudable, on other occasions ichorous, and as if mingled with decomposed blood and with dissolved tissues.

Thorough contamination of the blood and other fluids was invariably evidenced by fœtor during life, and by rapid putrescence of the body after death. In the heart and vessels the blood was fluid, there being but little fibrinous coagulum deposited, whilst there was tumefaction and softening of the spleen, together with general imbibition of the tissues. The ecchymoses already alluded to, conjointly with similar ones in the mucous membranes of the digestive organs, seemed, in like manner, to denote decomposition of the blood.

The order of sequence of the several organic changes was tolerably uniform in the different cases, appearing to deviate only according to the way in which the distemper had been contracted. Glanders seem to be conveyed from the horse to man in a twofold manner, namely, by *infection* and by *inoculation.* We are altogether in the dark as to the conditions which govern the former mode of propagation. Does abiding in the atmosphere of the diseased animals suffice?—is it that their exhalations operate upon injured portions of the skin, or upon the uninjured surface of the respiratory mucous membrane? In some of the cases there was no evidence of the morbid animal matter having come in contact with wounds of the skin in persons attacked. At present, therefore, we are compelled to assume general infection, apart from contact, inasmuch as the local affection on those occasions was invariably preceded by constitutional ailment, referrible to no other obvious cause, and nowise differing from any other violent eruptive fever. In the majority of cases, however, the disease was traceable to *inoculation* with the virus; the patients had either allowed abrasions of the skin to come in contact with the nasal secretions of horses they were tending, or had wounded themselves, whilst occupied in skinning or cutting up the dead animals. Here the disease dated from the moment of inoculation, and the progress of the supervening organic changes, could be pursued anatomically. Thus, when the finger had been injured, pain and swelling almost invariably set in, within a very brief interval, along the arm,—followed by red streaks and retiform spots along the skin. The lymphatic glands at the bend of the arm and in the axilla, became painful and tumid, and partially suppurated; in short, all the consequences of inflammation of the absorbents ensued.—Occasionally, the marks of phlebitis were apparent in the dead body. The original wound either turned to an ill-conditioned sore, or else healed up for a time, and then burst open afresh, on the period of incubation terminating, and the phenomena above described setting in.

However the disease may have originated, it makes very rapid progress, usually proving fatal within a fortnight or three weeks. It becomes chronic only when complicated with farcy, and contracted by inoculation from horses, labouring under like complication. The symptoms of farcy,—namely, cutaneous eruptions and ulcers, together with abscesses in the subcutaneous cellular tissue, and between, or in the substance of the muscles, then gradually spread from the injured part over the entire limb, and ultimately over the rest of the body. Glanders afterwards supervene, with concomitant fever, and the trains of disturbance above described. We may remark, that Rayer's researches have shown that simple farcy is equally communicable to man, and destroys life both in its acute, and in its chronic stage.

Both in glanders and in farcy, whether acute or chronic, it is sometimes no easy matter to determine, either from the history of the case or from the results of dissection, whether some of the appearances, as lobular pneumonia, purulent deposits within the lungs, muscles, joints, &c., be not rather due to simultaneous inflammation of the lymphatics and veins, with absorption of pus into the circulation, than to the original disease. Sometimes the returning vessels have been found perfectly healthy,—in a few instances decidedly inflamed,—whilst, in many, their condition does not seem to have met with due consideration and attention.

Very recently Engel (Oesterreich. Medic. Wochenschrift, 1842) has contended against the possibility of the transfer of glanders from horse to man, and sought to prove, by the aid of numerous skilful dissections of glandered carcases, that the disease is identical with tubercular mischief. The rise, progress, and termination of the local affection, the form of the ulcers, and many other circumstances, certainly offer many points in support of such an hypothesis.

# CHAPTER XI.

## CANCEROUS TUMOURS IN THE RESPIRATORY ORGANS.

The lungs are less prone than most other organs to cancerous disease. Indeed, pathologists of vast experience have encountered but isolated examples of pulmonary cancer.[1] As our acquaintance with tumours generally, and with malignant ones in particular, was, until lately, somewhat defective,—the diagnosis of a malady so rare as the present one, necessarily remained obscure, and the records, concerning its course, very imperfect. Several cases, recently observed by other pathologists, and some by myself, have enabled me to draw up a historical notice of the disease, which may possibly aid in its detection during life.[2]

Cancer of the lung is incomparably more frequent in males than in females. Out of 22 cases, collected by myself, 5 concern women, and 17 men. In childhood, the disease is unknown. Of the above 22 cases, 9 occurred between the 20th and 29th years; 8 between the 30th and 39th; 2 between the 40th and 49th; 2 between the 50th and 59th; and 1 between the 70th and 79th years. The morbid disposition is, accordingly, greatest in the prime of life.

The disease may be either *primary* or *secondary*,—more frequently the latter. But even where the cancer originally and mainly occupies the lungs, it is always deeply rooted in the organism, other parts being, simultaneously, more or less involved. This remark applies, particularly, to the secondary affection, in which the majority of the viscera, together with entire groups of lymphatic glands, often pass through every phase of carcinomatous degeneration, ere the lungs are assailed.

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[1] Out of 900 subjects examined, Bayle met with but one example of what he terms "Phthisie carcinomateuse."

[2] Besides the cases related by Morgagni, Bayle, Laennec, and Andral,—see Oettinger (Jahrb. d. Münchn. ärtzl. p. 98, Ver. 1835); Struve (Diss. de fungo Pulm. Lips. 1837); Heyfelder (Studien, &c. Bd. i, p. 62); Stokes (Diseases of the Chest); Carswell (Elementary Forms of Disease, fasc. iii, pl. 1, 2, 3); Durand-Fardel (Journ. Hebd. 1836, No. 33); Hope (Path. Anat.); Osius (Heidelb. Annal. Bd. vi, H. 1); Schwartz (Mittheil. Rig. Aerzte i, p. 131); &c.

In almost every instance, the tumours in the lungs were medullary; once only I met with the colloid variety. The medullary cancer was chiefly found in isolated masses; in the form of infiltration,—only where the lungs were the part originally affected. Primary medullaryi nfiltration is, as would appear from the few observations hitherto described with sufficient accuracy, characterized by one lung being exclusively involved; the other remaining exempt. The neighbouring lymphatic glands display the same species of degeneration, whilst other organs participate only in rare instances, and in a subordinate degree, as regards both size and development. The affected lung is either cancerous throughout, and the disorganization is nowhere distinctly circumscribed, but completely displaces the normal texture, from the apex to the base; or else isolated patches of healthy pulmonary cells may still be detected. The bronchial tubes vanish indeterminately in the encephaloid mass. The blood-vessels are partly compressed,—partly obliterated,—partly replete with the adventitious product;—the nerves are not traceable into the growth. The whole tumour presents an uniform, lardaceous structure, here and there pervaded by fibrous texture, and by darkish striæ and dots, corresponding to the amount of displaced pulmonary substance which may remain. In other instances the circumference of the morbid structure is firmer, and contains partly a white, nearly uniform, substance of medullary consistence, partly a pultaceous mass, which here and there discharges itself through the bronchi, leaving irregular excavations. The entire tumour is sometimes smaller than the lung, whose situation it occupies; at other times so voluminous as to displace neighbouring organs, and shove the heart towards the right side.

*Secondary cancer of the lung* assumes the form of isolated tumours, rather equably dispersed throughout both lungs,—superficially and deeply,—from the apex to the base. In a few instances the inferior lobes are described as having been principally affected. The parts of the body where the cancer originates and whence it spreads to the lungs, are very various. The bones and the testicles appear to furnish its most frequent starting point; and numerous examples tend to show that surgical operations for the removal of cancer in those parts, are very speedily followed by its transition to internal organs. Many instances are adduced in which the skin, and the mammary glands, the uterus, the liver, the membranes of the brain, the lymphatic glands, were the first assailed; I have seen a very remarkable case consecutive to primary cancer of the submaxillary gland. On the other hand, cancer in organs, whose veins are tributary to the portal system, does not appear to spread to the lungs, although it is known to lead very often to corresponding disease in the liver. This limitation of the disease to the capillary range of the portal vein, is the more remarkable, when we consider the promptness with which medullary cancer, in particular, proceeds from one cluster of lymphatic glands to others, situate in the remotest parts of the body.

It is obvious, from the foregoing remarks, that the lymphatics and veins are the conduits through which the cancer is transmitted from one organ to another, and thereby rendered constitutional. The veins have been clearly shown, by Langenbeck's interesting experiments, (Schmidt's Jahrbücher, vol. xxv, fasc. i, p. 90,) to take up cancerous matter. Injecting the veins of dogs with secretion from cancerous ulcers having produced mere lobular abscesses, the happy expedient suggested itself to Langenbeck, of injecting the fluid expressed from a non-ulcerated medullary tumour recently extirpated. A dog thus treated suffered intensely from dyspnœa, but survived, and was killed after a certain time. Its lungs were found to contain little tumours, identical in general properties, and also in respect of elementary cells, with those of the tumour in question. This affords no proof, indeed, that the veins are the original seat of cancer,—but it shows that they convey living cancer-cells, introduced into their canals, to the nearest capillary plexus, where, owing to their relative size, they become intercepted, and carry on the work of reproduction.

Secondary tumours within the lungs vary infinitely in magnitude, being found in the same lungs, as diminutive as hempseeds and as large as a man's fist; their average size is about that of walnuts. They are irregularly round, and the medullary display superficial indentations, while the gelatiniform growths are more uniformly spherical.

In the lungs, as in all other organs, medullary growths, not yet softened, have been found of very different degrees of consistency, and denominated accordingly steatomata, sarcomata, encephaloid masses; and, by earlier writers, occasionally, tubercles. In fact, I have sometimes observed them resembling a milky fluid, loosely suspended within cellular tissue, sometimes resembling the substance of the brain, at other times again to display a firm texture that grated on incision, or felt like hard sodden flesh. Their colour was alternately pure white, milk white, reddish white. A peculiar variety of medullary cancer is found, either partially or in totality, to contain a deposit of black pigment, and is called melanoma.[1]

Medullary cancer of the lung appears either to occupy, cell for cell, the place of the pulmonary texture, or else, in its progressive growth, merely to displace the adjacent pulmonary cells. In the latter case, and specially when situated near the surface of the pleura, or perhaps raised above its level, it is surrounded with a lax, cellular,—or a more firm fibrous capsule, which, by far-fetched analogy with the cerebral mass, have been likened to the pia and the dura mater. The direct transformation from the pulmonary substance is frequently demonstrable from the fact of true pulmonary vessels and minute bronchial tubes being traceable into their interior. Whilst, however, such bronchia, for the most part, remain stunted and compressed, those vessels, on the contrary, from the medullary tumour growing from within outwards, are preternaturally developed, of increased width, and possess very thin coats. *Gelatiniform cancer* is transparent, of a reddish yellow hue; its relation to the pulmonary structure is precisely the same as that of medullary cancer. It is, however, of still more exuberant growth. *The softening of the tumours* seems, in the majority of cases, to proceed from the centre, and very seldom from the circumference. The firmer tumours contain small excavations, filled, sometimes imperfectly, with a little limpid fluid, and with a crumbling substance. The layers immediately surrounding these cavities are friable, and of a deeper yellow than those beyond. The softer tumours present general softening of their entire mass, which is frequently transformed into a reddish white, or brownish pulp, mingled, in many instances, with blood derived from the softened and torn vessels. Gelatiniform cancer would seem to liquefy thoroughly and with great rapidity. From the relation to the pulmonary texture above mentioned, the soft cancerous substance soon finds an outlet through the air-tubes, and is excreted in the shape of forked sputa, resembling a white, or reddish brown porridge. Medullary cancer of the lungs being, however, for the most part, the reflex of more advanced degeneration in other parts, death commonly ensues ere the tumours have undergone softening. In the vicinity of the cavities described the pulmonary texture is, in part, mechanically compressed, in part condensed by inflammatory hepatization.

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[1] See cases by E. Schilling (Diss. de Melanosi, Francof. a. M. 1831); R. Froriep (Encyclop. Wörterb. vol. xiii); Carswell (l. c. fasc. iv, pl. 1, fig. 2), &c. The cases are quite distinct from those of simple diffuse deposition of black pigment within the lung.

The adventitious growths are sometimes rapidly, sometimes slowly, developed. Accordingly there are examples of the embarrassment to respiration lasting half a year,—others again of its preceding death by only a few weeks. The development and progress of the tumours are often quite latent; upon the whole R. Froriep's four stages (Berl. Encycl. Wörterb. vol. xiii) are rather anatomically correct than symptomatically definable. Occasionally, these growths, more especially if situate near the pleura, are developed amid inflammatory symptoms. Here the rise and progress of the disease are analogous to that of acute tubercle. The lung,—and for the most part also the pleura,—display a multitude of little tubercular bodies,—in some places no bigger than millet seeds,—equably distributed throughout textures replete with blood and serum, and rather softened. Were it not for the contemporaneous appearances in other organs, it would be difficult to distinguish these from real tubercles.

The pleura is often found studded with medullary tumours,—the bronchial and mediastinal glands in the last degree disorganized. Some of these glands often attain the size of hen's eggs, and press in various ways upon the lungs, the great vessels, and particularly the œsophagus. The interior of glands, infiltrated with cancerous matter, is occasionally in a state of softening, so that they constitute extensive cysts, replete with a brownish fluid. Softened medullary substance in the lungs sometimes breaks through ruptured vessels, comprised within its structure, into the interior of the pulmonary veins, where it goes on vegetating, until it reaches the left auricle of the heart. (See Heterologous Growths.)

Cancerous disease of the lungs never coexists with pulmonary phthisis. In rare instances cancer is confined to the pleural cavity, and to the lymphatic glands of the thorax, embarrassing the respiratory function, by pressing against the lungs or air-tubes. Here the malignant growths either spread in the manner before described, along the lymphatic system, without gaining the lungs, or, what is more common, cancer of the mammary glands undermines the walls of the chest, and penetrates into the thoracic cavity.

We annex, in conclusion, the rare example of cancerous tumours evolved in different parts of the air-passages. The same relation holds here as in the lungs; to wit, the disease is secondary. In the lungs, however, the cancerous cells are generally disseminated through the agency of the returning vessels; whereas the air-passages are apt to become involved through direct contiguity. Thus cancer of the œsophagus not unfrequently extends to the larynx and trachea, now and then establishing openings betwixt the two tubes (Sachse,—Kehlkopfs, und Luftröhrenschwindsucht, p. 188, et seq.; Albers,—Gräfe und Walther's Journ., vol. xix.) In rarer instances external tumours about the neck enlarge, ulcerate, and eventually perforate the walls of the air-passage. Very seldom do cancerous growths originate in the larynx or trachea. (See Trousseau and Belloc's 18th case, and instances collected by Albers—Beob. auf. dem Gebiete der Pathologie, vol. i, p. 109, et seq.) Of primary cancer of the trachea I have encountered but one solitary example. A female who had suffered from violent cough with scanty, fetid, and often sanguineous expectoration, died hectic, without having manifested disease of the lungs. The trachea was found throughout dilated—its mucous membrane transformed into a thick, lardaceous, superficially rough, eroded crust, and its remaining layers changed into a grayish-white, tolerably homogeneous, indurated mass. The cartilaginous rings were destroyed, and the whole coalesced with the adjacent cellular tissue, so as to compose one coherent hard tumour. The neighbouring lymphatic glands alone were found cancerous at the same time. Carswell has drawn a medullary fungus of the bronchial tubes. (Fasc. iii, Pl. 2, fig. 8.)

# CHAPTER XII.

## FORMATION OF CYSTS IN THE RESPIRATORY ORGANS.

Serous and other cysts have as yet been observed only in the lungs and larynx, and even there very rarely. *In the lungs* their development is quite latent. It is only after having attained a certain magnitude that they irritate the surrounding textures, and are separated through the agency of inflammation and suppuration. Amid violent and repeated hæmoptysis, they pass into the bronchia, and are ejected ; after which the patient sometimes recovers. Cysts thus voided, however, do not always come from the lungs, but now and then emerge from the liver into the bronchial tubes. A solitary example is known (Bally, Journal des Connaiss. Méd. 1838, No. 7) of a hydatid, detached through inflammation, making its way into the pleural cavity, leaving, in the upper and middle lobes of the right lung, a cavity in communication with the bronchia,—and determining pneumothorax. Where cysts are, after death, found firmly adherent to the parenchyma of the lungs, they are, at most, as big as walnuts, and seated exclusively in the inferior lobes.[1] They contain a limpid fluid, and generally several smaller hydatids, for the most part lying detached within the mother cysts, sometimes protuberate beneath the inner surface of the sac,—after the manner described by Hodgkin, respecting the development and growth of hydatid sacs. These secondary hydatids sometimes perish within the mother cyst, their remains afterwards appearing in the shape of flocculi, or gelatinous pulp, or of spirally convoluted membranes. These cysts always have a *double membrane,*—the external possessing a fibro-cellular character, and being either simply agglutinated to the surrounding textures, or else attached by means of radiating bands (Laennec). This external membrane is susceptible of various alterations; it either becomes unusually thickened, so as seemingly to consist of several concentric layers, and assumes a yellowish hue, resembling that of the middle coat of the arteries, or else takes up calcareous particles, and changes gradually into a bony capsule. This membrane contains the vessels of nutrition for the hydatid sac, and these, when irritated, may greatly modify the nature of the contents, rendering them gelatinous, or even purulent. The second, or inner membrane, is thin and transparent, resembling serous membranes. Secondary hydatids would seem to be generated between the two membranes, the internal one appearing studded with little pellucid granules, which, on enlarging, break away from the parent-membranes altogether.

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[1] Nevertheless, Andral once found nearly the whole of the inferior lobes of both lungs transformed into *capacious sacs* (Clin. Méd. t. iv, p. 386.)

I have had repeated opportunities of ascertaining that the acephalocysts (taken from various organs of the body) are the seat of real entozoa, namely, of the ecchinococcus hominis.[1] These parasites are imbedded in a grayish, transparent, smeary mass, on the outer side of the internal membrane of the sac, for the most part in clusters, and frequently in vast numbers. If the examination be long delayed, after dissolution has taken place, we meet with but solitary hooklets of the coronet of hooklets of these entozoa,—and this is also the case in the shrivelled cysts, with pulpy yellowish contents, mingled with membraneous debris.

Mohr's case of *hair cysts in the lungs* (Berlin. Med. Centralzeitung, 1839, No. 13) is unique in pathology. In both lobes of the left lung were found two large cysts, communicating with each other, together with several smaller ones, formed by thick, albuminous membranes, and containing masses of reddish hairs, some three inches long. In the larger cysts, the hairs were found lying in loosely-twisted tufts,—in the smaller ones, growing from the membranes by distinct bulbs.

*In the pulmonary veins* we have, again, a solitary example, detailed by Andral (l. c. p. 392), of a multitude of acephalocysts seated in both lungs, within small branches of the pulmonary vein,—presenting abrupt dilatations and alternate contractions.

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[1] Compare Siebold's researches respecting the ecchinococcus (Chemnitz. de ecchinococco hominis, Dissertatio Halensis; and Lebert, in Müller's Archiv. 1843, fasc. iii; also, Forbes's Brit. and For. Med. Rev. No. xxxiii, p. 194, et seq.)

Hepatic cysts existed at the same time, as did likewise moderate hypertrophy, conjoined with dilatation of the heart. In all the known cases, the cysts were accidentally discovered, either in persons that had died of other diseases,—or where they had been the actual cause of death, by exciting secondary inflammation of the surrounding parenchyma. Cysts generally coexisted in other organs, especially in the liver. In two instances (Andral and Mohr) tubercles were simultaneously present in the lungs, whereas Rokitansky concludes, from numerous observations, that the two diseases are incompatible with each other.

Cystic growths upon the walls of the air-passages are extremely rare. They are always simple serous cysts,—never acephalocysts. Albers (Beob. im Geb. der Path. vol. iii, p. 98) twice met with fibrous cysts, with pulpy contents, beneath the perichondrium of the thyroid cartilage. A few examples of hydatid vesicles upon the vocal chords are also adduced by Albers (vol. i, p. 96). I have myself seen a pellucid vesicle as big as a grain of hempseed upon the left vocal chord.

Finally, we have to advert to cysts in the cellular tissue, adjacent to the respiratory organs. These often occasion extreme difficulty of breathing. They occur in every variety of form and size, and are totally distinct from the cysts peculiar to the thyroid gland. Cases have been collected by Fleury and Marchessaux (Arch. Gén. Août 1839). Their growth is, for the most part, very slow, and, unless they form an external swelling, completely latent. An attack of catarrh often suddenly causes them to enlarge—and to produce death by suffocation. Laennec narrates two examples of cysts engendered beneath the costal pleura: in this situation I have met with a shrivelled acephalocyst the bigness of a nut, in which, with the aid of the microscope, sundry hooklets of the ecchinococcus were detected.

# CHAPTER XIII.

## PSEUDO-MELANOSIS OF THE LUNGS AND BRONCHIAL GLANDS.

No organ is so frequently, or under so great a variety of circumstances, a receptacle for the deposition of pigment, as the lungs. It was long known, that, with advancing years, these assumed a dark or black colour. With regard, however, to the morbid deposition of pigment but little was divulged, save a few loose observations (that of Haller, for example) prior to Laennec's classifying it with other blackish tumours, under the denomination of melanosis. He believed melanosis to be analogous to cancer, or at all events to be an adventitious structure akin to tubercle, and made out four varieties: encysted,—and free circumscribed masses,—melanotic infiltration,—and free deposit upon the surface of membranes. Accordingly he regarded melanosis as a product of malignant disease, which, though originally firm and crude, gradually softened, destroying the implicated organ. He was also at great pains to discriminate between melanosis and the ordinary black pigment of the lungs. The researches of Breschet (Rev. Méd. vol. iv, p. 304) and of Andral, led to a modification of Laennec's view. Having ascertained, from chemical analysis, that the composition of melanotic parts does not materially differ from that of the blood, they concluded that the black colouring arose from blood extravasated, under peculiar circumstances, in both healthy and diseased textures. Black induration of the lungs, more especially, was regarded by Andral as a particular form of chronic inflammation of the lungs. To the four forms above mentioned, this author added a fifth, namely, fluid melanosis. Meanwhile a new hypothesis, explanatory of infiltration of the lungs with black pigment, was started in England, and this, from its simplicity, and the plausibility it derived from local circumstances, gained credence with many pathologists, including, to a certain extent, Andral himself. The black substance was held to be mere carbon, inhaled from the smoke and vapour of coal and oil, which, it was averred, accumulated in the lung, producing inflammation and destruction of its textures. The utmost amount of black infiltration was imagined to affect the lungs of those who worked in coal-pits, and it was termed accordingly *anthracosis*. In the 'Philosophical Transactions,' for 1813, p. 159, Pearson proved, by chemical analysis, that the black substance in the lungs differed materially from pigmentum nigrum,—that of the choroid membrane, for example;—inasmuch as the former was not acted upon by hydrochloric or nitric acid, and must consequently be composed of pure carbon. This fact was confirmed by several other chemists, particularly by Christison (Gregory, Edinb. Med. and Surg. Journ. vol. xxxvi, p. 389); Graham (on Charcoal in the Lungs, ibid, vol. xlii, p. 323); and in France by Rilliet (Arch. Gén. 3me série, vol. ii, p. 160.) But, although the deposition in the lungs is here shown to differ from black pigment in other situations, the reception of carbon into the lungs from without is by no means demonstrated. Were such the case, it would follow that all persons exposed for a length of time to an atmosphere charged with coal-dust, soot, or lampblack, must inevitably become the subject of anthracosis; a result not borne out by experience. W. Thomson has shown, from medical reports issued from the more extensive coal districts of England and Scotland, including those where the coal is blasted with gunpowder, that black infiltration is not dependent upon, or indeed particularly frequent among individuals employed in an atmosphere laden with carbon. (Med. Chir. Trans. vol. xxi, p. 340.) Heusinger (Ueber Anomale Kohlen-und Pigmentbildung, Eisenach, 1823) has promulgated a different explanation, and one more in keeping with the processes of organic life. He maintains that black deposits in different parts of the body depend upon a deficient elimination of carbon, and, in particular, of carbonic acid. He further believes that they, in a certain sense, compensate for such defective process,—being especially prone to form in organs which afford the natural outlets for carbon,—although other organs may be similarly predisposed by disease. How far this view is applicable to spurious melanosis of the lungs and bronchial glands, will appear in the sequel. It offers at least the greatest amount of probability, inasmuch as black infiltration is the almost unfailing concomitant of the reparation of pulmonary disease, and in a greater or less degree of senile atrophy of the lungs (see p. 306). It is, however, not so satisfactory in relation to those melanotic tumours of the lungs or other organs, which bear the stamp of malignancy. We ought, in my opinion, carefully to discriminate between these and spurious melanosis,—the black tint being but an accidental, and as yet unexplained appendage to the malignant (for the most part medullary) growth. We must, therefore, concur with Schilling (l. c.) and admit two forms of melanosis, the one innoxious, the other malignant,—the former purely and essentially local, the latter prone to become constitutional and contaminate every part of the organism.

In advanced age the lungs are wont, even when without any vestige of disease, to exhibit more and more of a black tint. The substance which imparts this colour is everywhere pent up in the cellular tissue, and even in the membranous parts of the lungs, without occasioning induration or other change in the parenchyma, and without appearing in distinctly circumscribed masses. In some instances the black substance occurs in extraordinary abundance immediately beneath the pulmonary pleura, where it forms irregular, superficial elevations, disposed like coarse network. Streaks and little heaps of the same kind are interspersed between the lobules. Thus the entire lung acquires, especially at the surface, a firmer feel.

*During the reparation of tubercle* the black discoloration is both frequent and conspicuous, and is not limited to any particular period of life. Andral witnessed it in a girl of 9 years. Black, indurated nodules of irregular outline, from the size of a cherry-stone downwards, are found distributed through the sound portions of the lung, but chiefly in the upper lobe of each. They mostly contain a nucleus of curd-like, or moist chalk-like tubercle; frequently, however, the mass is perfectly homogeneous, of cartilaginous hardness, and affords a glistening section. We are justified by analogy in regarding the above nucleus as the remains of tubercle, thoroughly pervaded with black pigment. Secondly, the apex of the lung, perhaps the greater portion of the upper lobe, is totally transformed into an almost cartilaginous black mass, in which not a vestige of pulmonary texture is visible. A few, often dilated, bronchial tubes, with blind extremities, permeate the adventitious structure,—whilst the greater number, like the pulmonary vessels, are entirely obliterated. These indurated spots always firmly adhere to the walls of the chest, and generally contain the heterogeneous remains of tubercular reparation. Thirdly,—either in the midst of the induration just described, or adjacent to the black nodule, are to be seen shrivelled, shut cavities, whose walls consist of black hardened texture, and whose interior is filled with a gray-black, smeary mass, sometimes interspersed with calcareous granules. Obliterated vessels and bronchi terminate in the vicinity. Fourth, and lastly, in certain rare cases, shut cavities, lined with a thin, but firm, black shining membrane, occur at the top of the lungs, in which situation slight traces of long extinct tubercular disease are perceptible. These cavities contain nothing but air, and are sometimes traversed by strong and very tight cords, attenuated towards the middle. I do not hesitate to regard all these changes as resulting from the reparation of tubercular mischief, because, in most instances of the kind, whether in the lungs or elsewhere, I have found unequivocal evidence of tubercular disease. This, indeed, was the only affection which could be deduced from the history of the case, as adequate to account for all circumstances present. Further, the black tint displays itself in the bronchial glands, almost under the identical forms and conditions above assigned. In other organs, on the contrary, the reparation of tubercle is associated with incomparably less of the black degeneration. Hence the black colouring in the lungs would appear to be intimately connected with the disturbance of the respiratory function during phthisis, and quite independent of the accidental introduction of extraneous matter. It is doubtful whether it is ever the sequel of a sustained sub-inflammatory condition of the pulmonary texture. At least there is no good proof that black pigment can be thus produced, apart from tubercular disease.

When viewed by the microscope this pigment is seen to consist of granules 0·0008 of a line in diameter, for the most part disposed in dense, irregular heaps, between cellular texture, and in a few exceptions inclosed within spherical cells (from 0·015 of a line in diameter downwards). Even with the microscope, its elements are distinguished from those of tubercle by a dark brown or blackish tint, although in form and size the two are quite identical.

# CHAPTER XIV.

## DISEASES OF THE THYMUS GLAND.

The thymus gland is seldom found organically changed; mere alteration of volume being the ordinary anomaly, and one which, of recent times, has attracted much attention. The study of the other morbid affections of this gland is fraught with extreme difficulty,—the observations, especially of the older pathologists, being, in general, superficial and scanty. This, however, is of no great importance, because most diseases of the thymus are but the sequel of irremediable constitutional ailments.[1]

Inflammation of this gland is, in point of fact, very problematical; the so-called suppurative inflammation being ofttimes neither more nor less than tubercular softening. Haugstedt quotes a case of inflammatory softening from Portal, and another from Mason, where the thymus-abscess is said to have opened into the trachea. Cruveilhier reports a case of tuberculous thymus as chronic inflammation of the gland. (Atlas d'Anat. Path. livr. xv.)

Becker and Haugstedt cite some 15 examples of different-aged persons, subjects of more or less general tubercular disease, in whom the thymus was found involved. It was for the most part considerably enlarged,—very firmly united with surrounding parts, and either converted by tubercular infiltration into a hardened mass, or else partially destroyed by tuberculous softening. In three or four instances calcareous concrements, probably resulting from the retrogression of tubercle, were discovered in the gland.

As tubercle, so likewise constitutional cancer, has been known to assail this gland. Becker relates an instance of the kind, and cites several from other writers.

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[1] The bibliography of this subject will be found in G. F. Becker's treatise (De Gland. thor. lymphat. atque de thymo, &c., Berol. 1826); and in Haugstedt's (Thymi descriptio, &c., Hafniæ, 1832.)

According to Haugstedt, the thymus gland attains its greatest magnitude at the completion of the first twelvemonth of life; it is commonly 2½ inches long, 1½ inch broad, and four lines deep,—and weighs about 250 grains. From this period, it is said to remain stationary until the eighth year, after which it progressively declines. There are, however, many examples of this gland having been found in the adult, considerably above those dimensions. Becker has adduced numerous cases of more or less marked hypertrophy of the thymus gland in both children and adults.

Kopp (Denkwürdigkeiten, &c., vol. i, Frankf. 1830) first attempted to connect this preternatural enlargement of the gland, and its pressure upon the trachea, with an alarming ailment peculiar to childhood, which he accordingly termed "thymic asthma." This notion was, however, combated by Caspari and Pagenstecher, who showed (Heidelb. Annal. vol. viii, fasc. 2) that the disease alluded to is often coincident with a natural—nay, even with a strikingly small—thymus; and, inversely, that a greatly enlarged gland does not necessarily produce those asthmatic symptoms. J. F. H. Albers has very ably advocated the latter doctrine (Beob. im Geb. d. Path. i, p. 63, 1836). Nevertheless, many a case of sudden death, under asthmatic symptoms, has accrued from hypertrophy of the thymus gland.

There can be little doubt that the fatal asthma in question arises from a variety of causes; sometimes, indeed, from enlargement of the thymus gland; sometimes from remote affections of the respiratory and circulating organs; sometimes, again, from the pressure of swollen lymphatic glands (see Hugh Ley, l. c.), or of the hypertrophied thyroid gland. It may, moreover, be of an essentially nervous and spasmodic character. In any case the enlarged thymus, for anatomical reasons, can only, under certain circumstances, exercise injurious pressure upon the windpipe. It is usually the great vessels, as the descending vena cava and the right auricle, which suffer, and may thus induce sudden death through hinderance of the return of blood to the heart.

# CHAPTER XV.

## DISEASES OF THE THYROID GLAND.

The pathology of the thyroid gland will remain obscure, so long as we continue in the dark touching its physiological import. We shall, here, briefly relate what is known concerning inflammation of the gland, and its various forms of enlargement, usually included under the term bronchocele, or goitre.

*Inflammation* of this gland is rare. It may attack the organ, either when healthy, or when enlarged by previous disease.[1] Its course is more frequently chronic than acute. Within a very brief interval the gland often swells considerably,—becomes very bloodshot, tense, and painful,—its texture softened and friable, assuming, first, a brown-red, and ultimately a dingy-gray colour. The morbid anatomy of this grade of inflammation is but imperfectly known; that of the suppurative stage has been more frequently observed, and more fully described. Either separate abscesses form, or else the entire gland is converted into pus. Under favorable circumstances, the abscess opens externally through the skin;—there are, however, examples of its obtaining vent through the œsophagus, and determining a protracted fistula of the gullet (Unger, Beyträge zur Klinik der Chirurg. vol. i), or of its discharging itself into the trachea, and producing death by suffocation (Meckel.) After evacuation of the pus, together with numerous shreds of dead cellular tissue, the tumour collapses,—the gland, on the side affected, shrivelling into a hard, cellulo-filamentous knot, which adheres firmly to the skin, and the surrounding parts. Sometimes the shrivelling of the one, gradually brings on wasting of the other lobe.

*Bronchocele*, or *goitre*, results from various kinds of disorganization of the texture of the thyroid gland. Several species have been enumerated,—three, however, seem adequate to the purposes of practical surgery, namely, vascular *bronchocele*, (called by von Walther,—Neue Heilart des Kropfes, 1817,—"*aneurismal*"), —*lymphatic bronchocele*, which, strictly considered, would require further subdivision,—and *encysted bronchocele.* This classification does not, however, altogether meet the present state of our knowledge concerning the degeneration of textures,—we shall, consequently, proceed to give a survey of thyroid tumours, as observed at Leipsic;—although that locality does not appear to be prolific in remarkable cases of the kind.

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[1] See Meckel (Abhandl. aus der menschlich. und vergl. Anat. 1806); Rullier (Dict. des Sciences méd., art. Goitre, p. 525); A. G. Hedenus (Tract. de Gland. thyr., Lips. 1822, &c.)

We shall first treat of *hypertrophy of the thyroid gland*, a simple enlargement of the organ, without appreciable change of texture. This variety is frequent, and for the most part inconsiderable,—but it implicates the entire gland, and thus, perhaps, occasions comparatively greater disturbance than a much more extensive tumour of another kind. Both lobes of the gland, and even the middle one, swell so as to encroach equably on each side, against the trachea and the vessels of the neck. Serious accidents may arise, where the lateral lobes enlarge posteriorly, and tend to form a girdle round the trachea. Simple hypertrophy of the thyroid gland is almost wholly confined to youth, and is frequent, about the age of puberty, in both sexes—more so, however, in the female. Alternations of increase and decrease are especially apparent in this kind of bronchocele,—enlargement being most conspicuous at the approach of the menstrual period. In rare instances it is congenital,—augmenting, after birth, in a manner highly prejudicial both to respiration and to circulation. Hedenus has presented to the museum of Leipsic a preparation, in which the enlarged thyroid gland of a new-born infant is seen engirding the whole tube of the trachea;—strangulation was the consequence.

*Melicerous degeneration* of the thyroid gland is one of the most frequent forms. It occurs at all ages, and is uniformly attended with intumescence. It may involve the organ in whole, or in part. In the former case, the component granules are found unusually and unequally enlarged, and transformed into separate cells, filled with a tenacious, viscous, jelly-like substance, of the colour of honey. The entire part is hard, nearly bloodless, and but loosely coherent with the surrounding parts. Where, as frequently happens, only certain portions are disorganized, these form spherical tumours, varying in size, and imbedded cleanly in the healthy structure. They present a brownish, or yellow colour, and the consistency of jelly, or of melted glue. Sometimes they appear as an opaque, reddish, soft, or even lardaceous swelling. In general but few blood-vessels are visible in this goitre, although it may now and then be associated with exuberant vascular growth.

*Vascular bronchocele* is of much larger size. All the blood-vessels are amplified,—the veins, in particular, forming very dense, capacious, often knotted plexuses, and the whole texture consisting, apparently, of a dense coil of vessels. The substance of the gland has almost entirely lost its granular character—it is flabby, and dark red. After death, the tumour collapses considerably, and can only be restored to its original size by artificial injection. The walls of the arteries and veins are attenuated; the dilated membranes of the vessels contain considerable clots, and capacious cavities are found filled with black, coagulated blood. Vascular bronchocele affects the entire gland, principally, however, one or other lobe. It occurs most frequently in females, after the prime of life, and is, like simple hypertrophy, marked by periodical augmentation and decrease. This general dilatation of blood-vessels must not be confounded with the exuberant vascular growth, termed fungus hæmatodes,—to which the thyroid gland is also subject.

*Cystic formation* within the thyroid gland, is one of the most frequent causes of goitre. It occurs both by itself and in conjunction with other kinds of degeneration, and constitutes the largest and most unsightly of all tumours. Cysts of every variety and size, either solitary or in congeries, are encountered in every part of the thyroid gland; an entire lobe, nay, the greater portion of the whole organ being sometimes engaged in cystic development. The surrounding texture is seldom healthy, being generally compressed, flabby, and bloodless. The neighbouring organs are much embarrassed; the cyst becomes attached to them,—causing them to waste away, whilst in rare instances it so presses upon the trachea as to produce suffocation. The cysts are, for the most part, isolated; occasionally, however, they abut one upon another, so as to form a single multilocular capsule. Here, as elsewhere, they are composed of two membranes—namely, an external filamentous, and an inner serous one. The external membrane is either smooth or sends forth bands which attach it closely to the rest of the texture; in many instances it partially, if not wholly, ossifies.

The sac contains a limpid fluid, or a number of secondary hydatids,[1] or again a jelly-like substance—but, more commonly, a whitish or yellowish, crystalline pulp, consisting almost wholly of cholesterine crystals, with phosphate and carbonate of lime. In some instances the cyst accidentally inflames and becomes atrophied; in others it gradually fills with earthy matter, and is transformed into a hard, calcareous nodule. Cysts occur in the thyroid gland in both sexes, and nearly at all ages; more frequently, however, in females, after the prime of life.

It remains to notice goitre arising out of the development of cancerous and other tumours in the thyroid gland. I have never met with tubercles in this organ. That the above-mentioned roundish melicerous tumours belong to gelatiniform cancer is improbable. Of the occurrence of medullary cancer of this gland no doubt can be entertained; it has been observed twice by myself, and by other pathologists also. It often promptly attains a large size, either embodying, or else compressing and displacing the neighbouring parts. It is attended with considerable vascular development—frequently with an exuberance of growth, resembling fungus hæmatodes. Sometimes it softens at the centre, causing rupture of vessels and extravasation of blood. Occasionally black pigment is accumulated within its texture,—more or less. Medullary cancer of this gland must not be confounded with that of the lymphatic glands of the neck, which often simulates goitre. The different varieties of bronchocele occur so often independently of, or only accidentally complicated with other diseases, that it is by no means an easy matter to establish any constant relation betwixt them.

Endemic goitre is frequently, though by no means necessarily, coupled with cretinism. The attempts to connect the diseases of the thyroid gland with those of the genital organs, have led to no definite result; and the averred relation between bronchocele and the scrofulous and tubercular diathesis is groundless: Sauter (Oesterr. Jahrb. N. F. vol. xx, fasc. 1) has observed that persons affected with extensive bronchocele, seldom or ever become subjects of phthisis.

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[1] Lieutand mentions a case where the trachea was perforated by one of these acephalocysts (tom. iii, p. 28.)

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THE END.

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